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COMMUNICATIONS

MICROPHTHALMOS*

BY

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A FEMALE child was born after it had completed the sixth month of intra-uterine life but before reaching the seventh month. It lived for thirty hours and then died due to prematurity. A post-mortem examination was made three days after death. It was found that the child had a supernumerary digit on the ulnar side of the right hand, that the eyeballs were deeply sunken into the orbits and were smaller than normal. Behind the pupils an opacity could be seen. No other congenital defects were found.

One eyeball was removed, fixed and cut in serial sections. Unfortunately, for various reasons, it was not possible to make a dissection of the contents of the orbit.

The eyeball was found to measure 8 mm. in antero-posterior diameter and on naked eye examination was observed not to be absolutely spherical.

Histology

Sclera.—The sclerotic is of average thickness and of normal structure. Muscular insertions into the globe can be seen. In the lower anterior part of the eye there is a bridge of scleral fibrous

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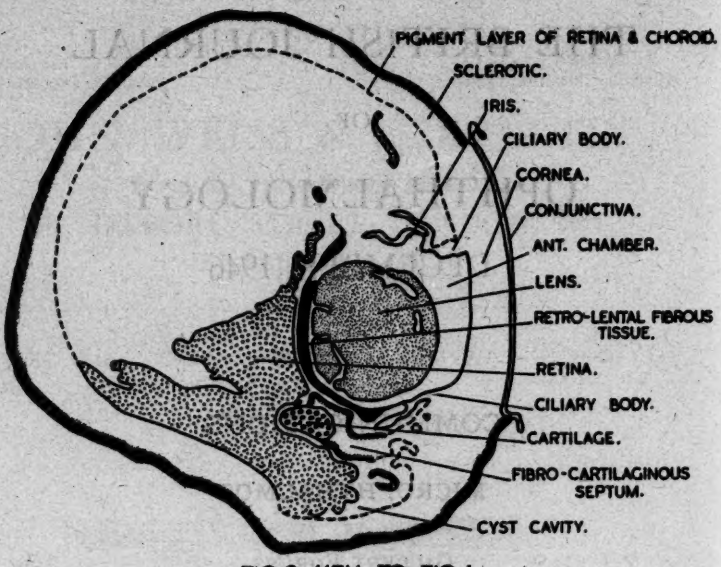

FIG. 2. KEY TO FIG. 1.

tissue cutting the cavity of the eye into two parts, one large and one small (Figs. 1 and 2). This smaller part is the reason for the asymmetry mentioned above and if it were amputated the section of the eye would be much more nearly circular. The bridge is of rather finer and more vascular fibrous tissue than the sclera proper, and in it portions of hyaline cartilage can be seen. This partition arises near the sclero-corneal junction anteriorly. It is apparent therefore that there is a cyst attached to the lower anterior part of the eye,

Cyst.—This is lined by choroid and by the pigment layer of the retina in its anterior one third. The choroid is thrown into irregular folds which protrude into the cyst. The pigment epithelium, which is present only on those parts which are covered by choroid, is very heavily pigmented and is irregular in depth. The appearance suggests that the pigment layer has been thrown into secondary folds on the folded choroid. The cyst is not completely cut off from the cavity of the eye and fragments of disorganised retinal tissue can be seen passing through the gap. The interior of the cyst contains a mass of plicated disorganised retina, in some places opposed to the choroid, in others to the fibrous wall of the cyst, and also lying apparently free in the cyst cavity. The outer wall of the cyst is continuous with the sclera. In places

FIG. 1.

Section of eye.

it appears to be of the same consistence as the sclerotic, and in other places it is much less dense. This suggests that the wall was imperfect and has been reinforced by condensed orbital fibrous tissue.

Conjunctiva and cornea.—The structure of the conjunctiva is normal. No clear Bowman's membrane can be seen in the cornea. The superficial corneal layers are unduly cellular and the fibrous strands are less dense and less homogeneous than in normal corneal tissue. The structure of this part corresponds to that of the sclera and is vascularised. Descemet's membrane and the epithelium covering it are normal.

Choroid.—The choroid lines the interior of the globe from the upper ciliary body to the opening into the cyst where it ends abruptly. The anterior one third of the cyst is lined by choroid of irregular depth and thrown into many folds. An irregular broken semi-circle of what appears to be vascular fibrous tissue passes from one free edge of the iris to the other behind the lens (Figs. 1 and 2) separating it from the disorganised retina. The pigment of the choroid is scanty. In sections beyond the periphery of the cornea and iris the lens can still be seen, and the choroid is found to line the whole of the inside of the sclera, including the inner surface of the fibro-cartilaginous partition separating the cavity of the cyst from that of the eye proper. There is of course a break in the continuity of the choroid where the retina passes out into the cyst and this gap can be clearly seen in peripheral sections. Beyond the gap the retina is directly opposed to the inner surface of the sclera. In the anterior part of the eye, the choroid is thrown into folds and it projects as vascular papillae into the interior of the globe.

Ciliary processes and iris.—These structures are small and poorly developed but the ciliary process on the side nearest the cyst shows an hypertrophied irregular outgrowth projecting backwards. The ciliary muscle is present but the muscles of the iris cannot be identified. The canal of Schlemm was not found. The iris appears to be turned backwards behind the lens and, as already mentioned, a bridge of vascular fibrous tissue passes between the free margins of the iris behind the lens.

Lens.—The lens is roughly spherical in shape and shows irregular projections here and there. No suspensory ligament can be identified. In the serial sections the lens disappears from the picture after the iris. The reason for this may be that the lens is abnormally large, or has prolapsed into the posterior chamber, or that there is a gap in the iris corresponding to that in the sclera and choroid. The superficial layers of the lens show some fibrillar

structure but the interior appears to be composed of structureless homogeneous vacuolated material. In other words there is a rind of more or less normal lens tissue with a degenerate cataractous centre.

Retina.—The pigment layer of the retina is opposed to the choroid from the ciliary body in the upper half of the eye round roughly one third of the circumference. The anterior half of the cyst is similarly lined. In the interior of the eye proper the retina is detached and crowded towards the lower anterior part of the posterior chamber (Figs. 1 and 2). In places there is some attempt at the formation of the normal layers but the rods and cones are not demonstrable. The detached retina is thrown into folds and it is difficult to be certain whether the layers of the retina are normally arranged or inverted relative to the choroid. In other parts the retinal tissue is completely disorganised. Where the choroid is absent within the cyst (*i.e.*, in the posterior two thirds of the sac) the retina is opposed directly to the cyst wall. The interior of the cyst contains imperfectly formed irregularly arranged retinal tissue. The ciliary part of the retina is detached and folded back behind the lens. On the side nearest the cyst this part of the retina is thrown into many complicated folds. Occasional masses of vitreous humour can be seen intermingled with retinal débris.

Comment

This was at first thought to be a case of pure bilateral microphthalmos but, as in so many of these cases, histological examination has demonstrated the presence of a cyst attached to the lower anterior part of the eye. The cyst is in fact quite small and was not obvious on macroscopic examination. It is quite common for congenital defects of the eye to be associated with deformities in other parts of the body. Swan *et al.* (1943), Swan (1944) and Gregg (1941) have pointed out the frequency with which congenital cataract is associated with congenital defects of the heart, and an abnormality of the limbs as an added defect is not uncommon (Mayou, 1904). Apart from abnormal topography the tissues are imperfectly developed. Vascularisation of the cornea has previously been referred to by Cruise (1905), the presence of hyaline cartilage in the sclerotic by Collins (1897), the hypertrophy of the ciliary body on the side of the eye next the cyst by Mayou (1908), and the failure of development of the rods and cones in the outer layer of the retina by Collins (1897). In many of the cases previously reported, the lens has been abnormal in shape, often tending to be spherical, with a degenerate centre, containing

vacuoles and inclined to prolapse. In this case the lens shows all these features and is undoubtedly cataractous.

Normally the foetal fissure in the human embryonic eye begins to close at the 11 mm. stage. In this case things must have begun to go wrong after invagination of the primary optic vesicle otherwise a congenital cystic eye would have resulted, no lens would have developed and differentiation of the retinal layers would not have occurred. Furthermore, defective development must have been initiated before closure of the foetal fissure. The critical period must have been between the 7 mm. and the 14 mm. stages, or in other words between the fourth and sixth weeks of intrauterine life. Clearly, in this case, there has been a failure of closure of the foetal fissure at its anterior end. Through this gap the inner layer of the invaginated optic cup has protruded to form a small cyst. Mesodermal condensation has occurred round the deformed vesicle, including the cyst, to form a scleral coat, and from this a septum has grown backwards in an attempt to segregate the cyst from the cavity of the eye proper. This septum is incomplete and is partly composed of cartilage. The choroid is absent over the area of eversion. According to Ida Mann (1937), there is a close association between the presence of the pigmented layer of the retina and the development of the choroid. The everted portion (retina) represents the inner layer of the optic cup, no eversion of the outer layer (pigment layer) taking place. One would therefore not expect choroidal development between the sclera and the retinal contents of the cyst.

The condition is certainly not an ectatic coloboma. If it were there would be no fibrous septum partially separating the cyst from the cavity of the eye, and the cyst wall would be lined by an undifferentiated, flattened, atrophic neural layer arising from the apposition of the two layers of the optic cup.

The layer of vascularised connective tissue passing from the free edge of the iris behind the lens corresponds to the condition described by Terry (1945).

Developmental abnormalities of this kind are attributable to a variety of causes. It is common ground that, whatever the noxious agent responsible, it must act early in embryonic life—in fact, during the first six weeks of pregnancy. The abnormality may be intrinsic to the embryo, *i.e.*, due to a defect of the chromosomal or genic pattern, and this explanation is said (Ida Mann, 1937) to account for hereditary and familial abnormalities and often for sporadic cases also. Again the causal factor may be a toxin transmitted through the placenta from the maternal circulation. Lastly, mechanical pressure or trauma to the embryo

may be responsible. In this particular instance the abnormal influence must have acted so early that it is not conceivable that mechanical factors could have been responsible. The history of the pregnancy and the family history were investigated. The parents are perfectly healthy, normally developed people. They have had three children, including the child which forms the subject of this paper. No congenital defects are present in either parent or in the two surviving children, and there is no history of deformities of any kind on either side of the family. It appears, therefore, that this case represents a sporadic developmental defect in an otherwise healthy family. The mother has not previously aborted or been delivered of a premature child. During her last pregnancy her health was undisturbed. According to the work of Swan *et al.* (1943), Gregg (1941) and Swan (1944) there appears to be some sort of association between congenital defects in general, and of the eye in particular, and the occurrence of an attack of rubella in the mother during the early weeks of pregnancy. In most of the cases reported by these authors the child was born prematurely, was puny, difficult to feed and in many cases only survived its birth for a few days or weeks. The most frequent ocular defect found was congenital cataract, and the suggestion is advanced by Hurst (according to Swan, 1944), that, because of the association of congenital cataract and developmental defects of the heart, the toxin affects the embryonic vascular system and damage to the hyaloid artery is responsible for the cataractous condition of the lens. Damage to the eyes is unknown in rubella, but the theory is advanced that the virus responsible need not necessarily cause the same manifestations in the developing foetus as in the adult. In view of this work careful enquiries were made as to the possibility of the mother of this child having suffered from any of the exanthemata during pregnancy. There was no history of any sort of illness during this time. It would appear, therefore, that this case originated either from a genetic defect, or that the mother suffered a sub-clinical infection during the early weeks of pregnancy which passed unnoticed but left its mark on the foetus.

Summary

1. A case of microphthalmos with cyst is described.
2. The embryology and aetiology of the condition are briefly discussed.

I wish to express my gratitude to Professor Ida Mann, D.Sc., F.R.C.S., Margaret Ogilvie Reader in Ophthalmology, Oxford,

for her helpful criticism and advice during the preparation of this paper.

REFERENCES

1. COLLINS, E. T. (1897).—*Trans. Ophthal. Soc. U.K.*, Vol. XVII, p. 254.
2. CRUISE, R. R. (1905).—*Trans. Ophthal. Soc. U.K.*, Vol. XXV, p. 332.
3. GREGG, N. MCA. (1941).—*Trans. Ophthal. Soc. Australia*, Vol. III, p. 35.
4. MANN, IDA (1937).—Developmental Abnormalities of the Eye. Cambridge University Press, London. Pp. 16 and 98.
5. MAYOU, M. S. (1904).—*Trans. Ophthal. Soc. U.K.*, Vol. XXIV, p. 340.
6. MAYOU, M. S. (1908).—*Trans. Ophthal. Soc. U.K.*, Vol. XXVIII, p. 107.
7. SWAN, C., TOSTEVIN, A. L., MOORE, B., MAYOU, H. and BLACK, G. H. B. (1943).—*Med. Jl. Australia*, Vol. II, p. 201.
8. SWAN, C. (1944).—*Jl. Path. and Bact.*, Vol. LVI, p. 289.
9. TERRY, T. L. (1945).—*Jl. Amer. Med. Assoc.*, Vol. CXXVIII, p. 582.

ADHESIVE EPISCLERAL REACTION IN THE OPERATIVE TREATMENT OF RETINAL DETACHMENT*

BY

L. WEEKERS

LIEGE

THE various procedures used in the operative treatment of detachment of the retina aim at causing adhesive choroiditis at a spot corresponding exactly to the place of the tear: the tear is to be engulfed by the area of the operative chorioretinitis and thus indirectly obliterated.

We believe that in addition to the choroiditis, one should endeavour to promote an adhesive episcleral reaction, as this contributes greatly to the immediate operative result and helps to preclude relapses.

1.—Experimental observations

We would first briefly recall certain experimental observations which we published previously: [1 2 3]

Fig. 1 refers to a simple but significant experiment. We used a cataract knife, considerably thinned by wear; the point was sharpened as much as possible so that the blade was but a thin flat needle, extremely sharp. After having instilled a few drops of cocaine, the point of the knife was driven into the eye of a rabbit at a point distant from the limbus, adjoining the *superior rectus*,

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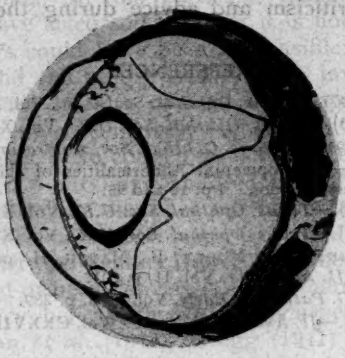


FIG. 1.

An experimental episcleral adhesion (rabbit).

the muscle itself being seized by forceps to fix the globe. The knife penetrated through all the layers: conjunctiva, episclera, sclera, choroid, and retina, but is driven only just far enough for the perforation to be complete.

Immediately afterwards, the place of the perforation can readily be observed with an ophthalmoscope; it shows as a small white spot; sometimes a slight haemorrhage may be noticed. Later, there is discrete pigmentary reaction.

After a variable length of time, the eye is enucleated and put into preservative fluids (fixing agents and alcohols). In the normal rabbit eye so treated, the retina becomes detached. When, however, a preliminary scleral perforation is performed, even though only a short time before (3 or 4 days), the retina, detached everywhere else, remains attached at the point of perforation (Fig. 1).

Where several perforations are performed, a few millimetres apart, the retina remains adherent at each spot. Fig. 2 shows four such adhesions.

Histological examination shows the mechanism of these adhesions. The scleral gap resulting from the perforation heals with the aid of the episcleral tissue. This proliferates, penetrates into the wound, develops in depth, and reaches the retina. The newly formed episcleral tissue and the retina become firmly adherent. For the section shown in Fig. 3 enucleation was carried out six weeks after experimental perforation. As a result of the retraction of the tissues, the episcleral tissue is artificially detached from the sclera, but the firmer adhesion to the retina remained intact.

To test whether the episcleral reaction noted in rabbits also takes place in the human eye with its substantially thicker sclera, human

eyes were perforated at various times before enucleation necessitated by different diseases. It was found that in the human eye too, even when the scleral gap is very narrow, the proliferating episcleral tissue reaches the retina within a few days.

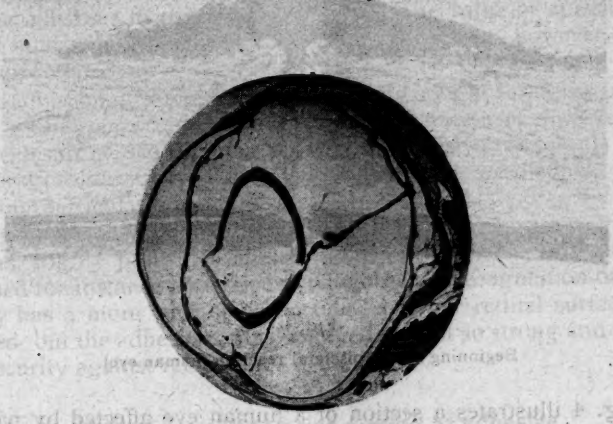


FIG. 2.

Four experimental episcleral adhesions (rabbit).

FIG. 3.

Adhesive episcleral reaction (rabbit).

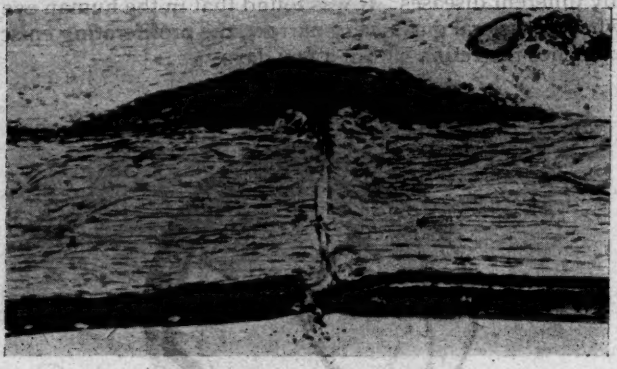


FIG. 4.

Beginning of an episcleral reaction (human eye).

Fig. 4 illustrates a section of a human eye affected by painful absolute glaucoma. Five days before enucleation the eye was perforated as in the experimental animal. Histologically, considerable proliferation of the episcleral tissue is seen, extending into the scleral gap which is still mainly empty. After about 8 days adhesion between the episcleral tissue and the retina can be seen.

These and other experiments allow the following general conclusions:

1. Wounds and defects of the sclera heal by rapid and active proliferation of the episcleral tissue. Healing in the sclera itself is slight or negligible.

2. In a perforating scleral wound, however narrow the perforation, the newly formed episcleral tissue proliferates rapidly in depth, and becomes firmly adherent to the retina—a point of significance in the operative treatment of retinal detachment.

2.—Perforations of the globe in the operative treatment of retinal detachment

Whether diathermy, electrolysis, galvanism or chemical cauterization is employed, the operation for retinal detachment usually involves one or several perforations of the globe.

Gonin used thermocautery to perforate the eye in order to reach the retina and occlude the tear. Weve, at first, used the diathermy needle and is still of opinion[4] that, in certain cases, it is advantageous to coagulate the retina directly. In contrast, multiple diathermic microperforations (Weve, Safar) aim at producing an

adhesive choroiditis. Perforation of the globe in the course of the operation also allows the escape of the sub-retinal fluid, and it is generally agreed that this evacuation should be as complete as possible in order that the detached retina may be better placed to become adherent to the wall of the eye. The value of episcleral-retinal attachments that result from perforation are, however, not fully appreciated.

The adhesion of the retina which is being sought for at the place of the retinal tears and the consequent obliteration of these tears, are the result of two different processes: adhesive choroiditis and episcleral proliferation. Though associated, they are distinct, each affording particular advantages.

Episcleral proliferation gives the retina limited but strong adhesions, highly protective against relapses. Adhesive choroiditis obtained for instance by a superficial diathermic coagulation of the sclera, has a more wide-spread action; a larger retinal surface is reached, but the adhesions thus obtained are not so strong and give less security against relapses.

3.—Technique of operation aiming at adhesive episcleral reaction

An indispensable condition for obtaining episcleral retinal adhesions is that the eyeball be completely perforated. Only then can the newly formed episcleral tissue originating from the surface reach the retina.

Animal experiments showed that the episcleral reaction occurs whatever the mode of perforation (needle, knife, galvanocautery, diathermy electrode, etc.), but the reaction is the quicker the less the operative trauma.

Experimentally, the procedure which appears most efficacious consisted in simply perforating the eyeball by means of the point of a sharp cataract knife, but it has the drawback that it may cause a haemorrhage.

When a perforation is performed by means of the fine point of a galvanocautery, proliferation of the episcleral tissue towards the retina seems rather slower; it is delayed by the presence of necrotic tissue in the wound.

In the course of an operation for detachment there is a distinct advantage to perform several perforations so as to increase the number of episcleral adhesions. Therefore it is necessary to avoid escape of sub-retinal fluid after the first perforation, for this would soften the globe and make subsequent perforations more difficult and risky.

The following procedure is simple and satisfactory:

An ordinary angled and insulated diathermy needle (length: 2 mm., diameter: 0·15 mm.) is used. The diathermy apparatus was previously gauged on an eye to be enucleated; thus it was possible to determine beforehand the weakest current required for an easy penetration by the needle. The knob of our apparatus (Sanitas Ophthalmotherm) is turned to division 2. The point of the needle is placed against the sclera. Slight pressure applied as soon as the current is on causes instantaneous perforation. The needle is then swiftly withdrawn without turning off the current. The whole manoeuvre is carried out in less than a second. Generally, no fluid escapes through the perforation thus performed.

The reason for the electric current being turned on is two-fold: to facilitate perforation and avoid a haemorrhage. The aim of the preliminary gauging is to determine the minimum intensity of the current which ensures the practically instantaneous penetration of the needle into the sclera. Occasionally, when the first perforation is performed the current proves insufficient for an easy penetration of the needle; the intensity is then slightly increased for the subsequent perforations. These perforations leave ophthalmoscopically visible scars after cure: small white spots with a slight pigmentary reaction. They are similar to the scars observed in the experimental animal. According to the area to be covered, the number of perforations varies from three to twenty. It is remarkable that the last perforation can be carried out as easily as the first.

4.—Steps in the operation for retinal detachment incorporating puncture for adhesive episcleral reaction

1. We first apply the non-perforating diathermic electrode on the sclera in order to promote the indispensable choroidal reaction at a place corresponding to the retinal tear.

2. We then carry our perforation of the globe as described in detail above—not to promote choroiditis (which has already been obtained by the superficial electrode) but to induce participation of the episclera. The intensity of the current is just sufficient to facilitate the penetration of the needle and to avoid a haemorrhage. The needle is very thin; its penetration and removal last less than a second together; under those conditions there is no escape of sub-retinal fluid and the eyeball does not soften. As many perforations as required can be carried out without difficulty.

By means of the diathermy needle, both the choroidal and episcleral reactions can be induced simultaneously (*e.g.*, method of microperforations of Weve and Safar). We believe, however, that, in order to obtain the two reactions with utmost efficiency, it is best to promote them separately, first the choroidal reaction by means of the non-perforating superficial diathermy electrode, then the

episcleral reaction by means of the perforating diathermy electrode.

3. We conclude the operation by the evacuation of the sub-retinal fluid. This may be achieved by means of a perforating diathermy needle but, in order that the evacuation be thorough, which we consider as greatly desirable, it is necessary to use a needle sufficiently thick and an electric current sufficiently strong; this may be comparatively dangerous. We prefer to use the galvanocautery in the following manner, which appears to be the least disturbing.

The cautery is heated to a dark red colour. The sclera is then lightly touched at the required spot with the sharply bent point of the cautery, which is immediately withdrawn. Thus a small cup is formed. After an interval of a few seconds, the same manoeuvre is repeated and the cup deepened. With two or three such manipulations the perforation is completed and the fluid escapes. Sometimes, even when the perforation has been completed, the subretinal fluid does not escape, because the opening has been obstructed by some necrotic tissue. In such cases we insert the blunt end of a lacrymal probe into the opening; when it is withdrawn, the sub-retinal fluid escapes freely. A single perforation is sufficient for the entire evacuation of the fluid.

It may appear exaggerated to study minutely a surgical act so simple as perforation of the eyeball during operations on detachment. This is surely not the case. Operations on detachment call for no special manual skill, yet the results vary considerably with different operators. When choosing a method, it is not sufficient that it ensures cures; it is necessary that the proportion of cures be as high as possible, and with but few relapses. In operations on retinal detachment, perhaps more than in others, success depends on an accumulation of details and on full appreciation of the limitations and effects of the various steps employed.

The use of a simple diathermy needle offers a good illustration. Several factors (length and diameter of the needle, intensity and duration of the current) have to be taken into consideration, which may combine differently and produce entirely different effects. If the needle is very short and non-perforating, adhesive choroiditis may take place, but there will be no adhesive episcleral reaction. According to the way in which the diathermy needle is used, the retina itself will or will not be coagulated. Certain technical conditions enable many perforations without undue trauma; others diminish the risk of haemorrhage. According to the method employed, the evacuation of the sub-retinal fluid will be partial or complete.

These and such-like technical aspects influence the proportion of cures. Perforation of the eyeball in the course of operation of the retinal detachment deserves special study, and the value of the adhesive episcleral reaction to be more widely known.

Summary

The various procedures used in the operative treatment of retinal detachment aim at promoting adhesive choroiditis at the retinal tear. Based on experimental and clinical observations, and especially on operative results, the author believes that, apart from adhesive choroiditis, it is necessary to create an adhesive episcleral reaction, the importance of which is paramount, because it affords particularly firm attachments which militate against relapses.

An adhesive episcleral reaction takes place irrespective of the method of perforation provided the perforation is complete. The episcleral tissue proliferates actively, progresses in depth and becomes firmly attached to the retina. The author describes an operative procedure which gives a satisfactory adhesive episcleral reaction.

REFERENCES

1. WEEKERS, L.—*Arch. d'Ophtal.*, Vol. XXXIX, p. 583. 1922.
2. ———*Arch. d'Ophtal.*, Vol. LII, p. 636. 1935.
3. ———*Arch. d'Ophtal.*, Vol. II, p. 193. 1939.
4. WEVE, H. J. M.—In Modern Trends in Ophthalmology, ed. F. Ridley and A. Sorsby, London, Butterworth, p. 553. 1940.

SULPHAPYRIDINE-RESISTANT KOCH-WEEKS CONJUNCTIVITIS*

BY

H. J. STERN

(MAJOR, R.A.M.C.)

IN *The Lancet* of May 26, 1945, I published with B. Miterstein a report on the outstanding results obtained with sulphapyridine by mouth in Koch-Weeks conjunctivitis. Three grammes of sulphapyridine daily in adults and correspondingly less in children, without any local treatment, clears up the conjunctivitis completely in so short a time that the hitherto used treatment with silver nitrate seemed slow and antiquated in comparison.

* Received for publication, July 12, 1946.

In the above quoted paper we wrote :

> This treatment always gives prompt results. The swelling of the lids and the blepharospasm disappear on the second day and the abundant discharge abates; the eyes can be opened, the pain has subsided, and the patients report that sleep is again possible. Next day the discharge has sometimes stopped altogether, and the lids are normal, though the conjunctiva still shows some swelling and redness. This is a critical stage, since the patients are apt to discontinue treatment because they no longer feel any discomfort; but if treatment is stopped at this stage, the conjunctivitis often recurs, and sometimes takes weeks to cure.

The treatment outlined above has never disappointed. Its efficacy has been confirmed by Bland and Wilson (*British Journal of Ophthalmology* Vol. 29, No. 7, June, 1945) and others. Bland and Wilson came to the conclusion that two doses of 1·25 grammes given on two consecutive days are sufficient to cure all cases of Koch-Weeks conjunctivitis with very rare exceptions. This is certainly the case; and all the same it seems essential to continue treatment for 2 or 3 more days in decreasing doses to prevent relapses and, what is more important, to prevent the creation of sulphapyridine-resistant stains.

In our first publication we had shown that relapses after insufficient treatment do not respond to a second course of sulphapyridine, and a series of particular cases observed lately seems to confirm the reality of this danger.

During the past four years I had occasion to see a considerable number of East African negroes in military hospitals in the Middle East. The result of the treatment of Koch-Weeks conjunctivitis in these patients with sulphapyridine has been so disappointing in almost all cases that it had to be given up and the old-fashioned silver nitrate reinstated. This was all the more disappointing because Koch-Weeks conjunctivitis takes usually a particularly severe form in these Africans. Profuse muco-purulent discharge, marked chemosis and quite frequently the formation of extensive membranes make the clinical picture rather alarming. The disease takes, fortunately, however, always a benign course. All cases recovered entirely without complications.

It seems that this failure of sulphapyridine in East Africans can be explained with the existence of a sulpha-fast strain of the Koch-Weeks bacillus amongst these people. It seems, moreover, the only possible explanation as no case is known of individual or racial non-response to sulphanilamides in infection with organisms known to be sensitive to these drugs.

There exists a parallel in gonorrhoea of East Africans. This

disease, rampant in their homeland and relatively frequent in African troops in the Middle East, fails frequently to respond to routine sulphapyridine treatment. The reason for this seems to be that sulphapyridine-resistant strains have been developed amongst these people. The sale of sulpha-drugs is unrestricted in East Africa, and probably every sufferer from gonorrhoea has tried once or twice to cure his disease with the famous M. & B. 693. The outcome of this enlightenment of the African negro is, of course, that not only he fails to cure his gonorrhoea because of insufficient treatment, but also makes this task more difficult for the V.D. specialist whom he consults sooner or later. A similar state of affairs has been reported from French North Africa.

The possibility suggests itself of sulphapyridine-resistant strains of Koch-Weeks bacilli being harboured by carriers who themselves have only a mild chronic conjunctivitis but are in a position to infect others with it. This infection with a sulphapyridine-resistant strain would explain the failure of the otherwise most reliable treatment with sulphapyridine.

It might be possible to prove this hypothesis with bacteriological tests. The conditions in an isolated military hospital make this unfortunately, impossible. Ophthalmologists in a most favourable position might have the opportunity to do so if they come across similar cases.

POSTERIOR UVEITIS IN A CASE OF SARCOIDOSIS*

BY

FRANK R. NEUBERT

LONDON

A considerable literature on the syndrome of uveo-parotid fever, originally described by Heerfordt, emphasises the frequent occurrence of this disease, the widespread nature of its manifestations, an apparent difficulty in the diagnosis of some cases, and a certain lack of agreement among those writers who endeavour to designate the responsible agent.

A bacillus, allied to that of tubercle, is usually indicted, which shows a predilection for certain tissues of which the uvea, parotid gland, lung, skin and intestinal canal, appear to be sites of election.

The case under consideration is of exceptional interest in-as-much as the abdominal picture is probably unique and I am unable to find any published report of a similar condition in the eye.

* Received for publication, September 9, 1946.

The literature shows that there is little specific reference to involvement of the cornea. Keratitis (when present), is said to be interstitial in character, as in the present case, affecting the lower part of the cornea first. Increase in the intra-ocular tension may produce an oedema which obscures it.

Iritis is the most common ophthalmological manifestation and two types have been described.

(*a*) Benign serous, which shows slight ciliary injection, fine posterior corneal precipitates, and a moderate number of cells in the aqueous.

(*b*) Nodular type which is extensive and accompanied by marked ciliary injection, oedema of the cornea, larger and more numerous posterior corneal precipitates, and many cells in the aqueous.

Opinions regarding implication of the posterior uvea and retina vary. Levitt states that it has been reported only by thirteen writers, and King states that, except in a case reported in a personal communication by Reis and Rothfeld, he is unable to find in the literature any case of involvement of the choroid or retina. After evidencing the extreme rarity of sarcoid as a cause of local chorio-retinitis, he suggests that it should be regarded as a possible cause in cases in which tuberculin tests are negative and syphilis has been negated. Berg states that choroiditis may occur.

Verhoeff found that in all cases of localized chorio-retinitis in an active stage, in which he examined the eyes histologically, the lesions were characteristic of tubercle. The chief features being an accumulation of epithelioid cells interspersed with a few giant cells and surrounded by a sparse infiltration of lymphocytes, but without necrosis or sclerosis (Duke-Elder).

Duke-Elder states that occasionally the posterior segment is affected also and whitish-yellow masses of histologically typical sarcoid structure may be scattered over the fundus. Levitt apparently considers this to be a periphlebitis, describing yellowish masses in the fundus with a tendency toward perivascular situation. Walsh reports one case showing numerous superficial discrete white spots in the paramacular area and normal vessels.

Reis, Rothfeld and others report that papilloedema, followed by optic atrophy due to involvement of the optic nerve may also occur.

Longcope and others state that the disease occurs often without attendant abnormalities of the skin.

In the series of cases of "Chronic Iridocyclitis and Tuberculosis" reported by Brooks, Juler and Williams, six had uveo-parotitis. Three of these showed a cutaneous tuberculide, maculo-erythematous in type, and three had no skin lesion, as in the case under discussion.

The literature contains only references to posterior uveitis in sarcoidosis and this is apparently the first case to be described and

illustrated. The history and clinical findings are therefore of considerable interest.

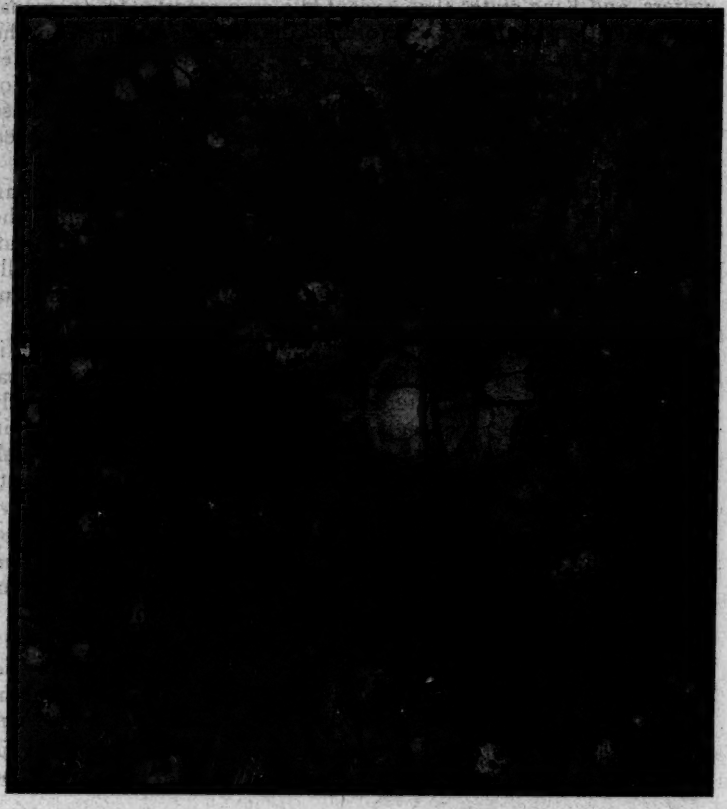
Case history

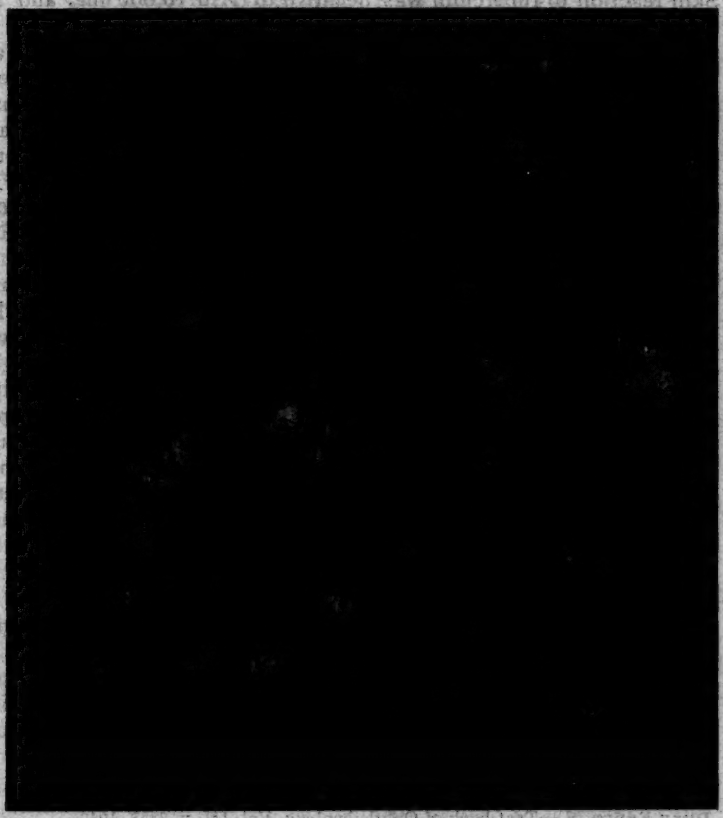
1. At the age of two years a radical mastoidectomy was performed for chronic mastoid suppuration. Nothing exceptional was noted in the hospital report.

2. At the age of seven he had malaria whilst living in India.

3. At the age of twenty-four he states that vision deteriorated rapidly after looking at a solar eclipse with the naked eyes. The vision was found to be:—R.E. 6/12, L.E. 2/60.

Both fundi showed coarse disseminated choroidal scarring, with some fresh patches, and slit-lamp examination showed interstitial scarring and cells in the vitreous.





Wassermann reaction was performed twice, and was negative.

Dental X-Ray was also negative, and an otological examination, on account of a history of aural discharge, disclosed merely the scar of a mastoid operation.

The patient was treated with mercury and potassium iodide and some months later vision was improved to,

R.E. 6/9 (with −0·75 sph) and L.E. 6/36.

4. In 1941 (aged 29 years) after severe overwork during the air raids on London he collapsed with a bad cough and loss of weight. Tuberculosis was diagnosed, and he spent five months in a sanatorium. Although the radiogram of the chest was typical of advanced miliary tubercle, the blood sedimentation rate remained at about 7 and the patient's general condition commenced to improve.

Skin reaction to tuberculin was negative to 1 in 10,000 mg. and 1 in 1,000 mg. but strongly positive to 1 in 100 mg.

During this time he had abdominal symptoms and developed a swelling. A tentative diagnosis of chronic appendicitis was made. An X-Ray examination of the intestinal tract showed constrictions of the caecum with a well-marked "string-sign," typical of regional ileitis. The caecum was resected and a microscopical examination of a section was typical of sarcoidosis.

5. General health improved and in August, 1943, his vision was R.E. 6/24, L.E. 6/24, correcting with glasses to R.E. 6/6 and L.E. 6/12.

At the examination in 1943, and again in 1944, when the fundus drawing was made, no evidence of anterior uveitis could be seen with the corneal loupe, but microscopic examination could not be made.

The fundus changes were not quite typical of "old choroiditis" none of the fibrosed areas showed a glistening white sclera and only the small peripheral ones appeared whitish. The number and size of the lesions decreased centrifugally. The scattered pigment was a deep sepia and was punctate in form. On two areas this pigment took the form of "wheat-sheafs" which I have attempted to depict. Although most of the pigmentation was beneath the vessels, some appeared to be over them.

Conclusion

A case is reported of disseminated retino-choroiditis in a case of sarcoidosis of the lungs and alimentary canal.

I am indebted to the Secretary of the Manchester Ear Hospital for tracing a case history of 30 years ago; to Dr. Werner of Dublin for particulars from his father's case histories; to Dr. Morland for clinical details of the chest and abdominal condition.

BIBLIOGRAPHY

DUKE-ELDER, S.—Text-book of Ophthalmology, Vol. III, p. 2358, 1940.

JERSILD, M.—Syndrome of Heerfordt (uveo-parotid fever), manifestation of Boeck's sarcoid *Acta Med. Scand.*, Vol. XCVII, pp. 322-328, 1938.

KEMP, R.—Note on chronic irido-cyclitis, with special reference to sarcoidosis of Boeck. *Brit. Jl. Ophthal.*, Vol. XXIII, pp. 455-460, 1939.

KING, M. J.—Ocular lesions of Boeck's sarcoid. *Trans. Amer. Ophthal. Soc.*, Vol. XXXVII, pp. 422-458, 1939.

KINDT, P.—Three cases of nodular iritis (iritis Boeck?). *Acta Ophthal., Kbh.*, Vol. XVIII, pp. 38-50, 1940.

LEVITT, J. M.—Boeck's sarcoid with ocular localization; survey of literature and report of case. *Arch. Ophthal., Chicago*, Vol. XXVI, pp. 358-388, (Sept.) correction p. 175 (Oct.). 1941.

POE, D. L.—Sarcoid of external ear. *Ann. Otol., etc., St. Louis*, Vol. XLIX, pp. 771-775, (Sept.). 1940.

WAGENER, H. P.—Ocular lesions in sarcoidosis of Boeck. *Amer. Jl. Med. Science*, Vol. CCIII, pp. 300-309. 1942.

WALSH, F. B.—Ocular importance of sarcoid; its relation to uveoparotid fever. *Arch. Ophthal., Chicago*, Vol. XXI, pp. 421-438. 1939.

WYCKHAM, W. D., JULER, F. A., WILLIAMS, E. R.—On the relationship between chronic iridocyclitis and tuberculosis, and the appropriate therapy. *Brit. Jl. Ophthal.*, (July). 1940.

POSTERIOR RING ABSCESS OF METASTATIC ORIGIN IN BEHCET'S DISEASE*†

BY

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JERUSALEM

A POSTERIOR ring abscess of the cornea of non-traumatic origin seems to be a relatively rare condition. In the case described below this condition was a complication in a patient who was suffering from the so-called Behcet syndrome. Elsewhere the writers discussed this case along with a number of similar cases seen by them, under the title: "Behcet's disease as manifestation of a chronic septic condition connected with a constitutional disorder." The syndrome consists of the following main symptoms: aphthous stomatitis, general ulcers, uveitis (recurrent iridocyclitis with hypopyon), retinitis, optic neuritis and erythema nodosum-like skin eruptions on the lower extremities. It was emphasized by the writers that the syndrome in question represents a chronic septic condition produced by staphylococcus aureus and that this peculiar kind of sepsis (best characterized as sepsis lenta), may only occur in a constitutionally predisposed individual whose power of resistance is weakened, in most cases by latent tuberculosis.

As the corneal lesion in question was merely touched upon in the paper previously mentioned, which was primarily intended to elaborate the pathological background of the syndrome including the ocular disturbances involved, it seems to be worth while dwelling in somewhat greater detail on this complication.

R. R., male, aged 28 years, since October 23, 1944, under prolonged observation and treatment for recurrent uveitis and retinitis, again presented himself on February 13, 1945, complaining of unbearable pain in his left eye, caused by an exacerbation of the disease. An additional complication in the form of a posterior ring abscess had made its appearance. The patient was in a state of prostration. His temperature was 38·7°C. On the scrotal skin two ulcers were present, their surface covered with purulent secretion.

Eye examination.—R.E.: Visual acuity amounted to 5/18. Cornea and aqueous humour were clear, the pupil was well dilated and in the retina small foci along the vessels accompanied by small haemorrhages were seen. L.E.: totally blind for two months, showed severe ciliary injection, chemosis of the bulbar conjunctiva, oedema of the corneal epithelium and an annular infiltration of the posterior wall of the cornea, the ring being open in its lower part

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from 5 to 7 o'clock. A portion of the cornea approximately 2 mm. wide was left clear between the infiltration and the limbus (Fig. 1). The anterior chamber was shallow and the aqueous humour cloudy with no visible hypopyon formation. The pupil, as far as could be seen, was partially dilated wherever not impeded by posterior synechiae. A complicated cataract was present and the ocular tension increased.

The day after admission to the hospital the annular infiltration advanced into the anterior layers of the cornea, especially from the

FIG. 1.

temporal side. In order to relieve the tension a paracentesis was performed. Culture of the aqueous humour showed a growth of staphylococcus aureus. The following day the annular infiltration did not change noticeably and ocular tension was still at the upper limit of normal. The chamber was reopened daily during the following days and the cultural findings of the aqueous were repeatedly positive. Staphylococcus aureus was also present in the two scrotal ulcers, while the blood culture proved sterile although temperature had risen up to 38·8°C.

Treatment was started with sulfathiazole, 3 grams a day, for a week's time only when it had to be stopped because of general malaise and severe vomiting of the patient. In spite of this course with sulfathiazole the aqueous humour continued to show a growth of staphylococcus aureus and the annular infiltration remained unchanged.

On February 27, the patient complained of intense headaches and ran a temperature of 37·5°C. Blood culture, taken after careful cleansing of the skin, over the cubital vein, with iodine and alcohol showed staphylococcus aureus.

On March 1, systemic penicillin treatment was commenced. Every three hours, day and night, 12,500 O.U. were administered intramuscularily for a fortnight until the total dose reached 1,000,000 O.U. The second day after initiation of the penicillin injections the general condition of the patient improved, the infiltration of the left cornea lessened, the culture of the aqueous humour for the first time became sterile and remained so in the consecutive examinations. Even the scrotal ulcers, which in culture continued to show staphylococcus aureus, grew smaller and became flatter and cleaner. On the seventh and the eleventh day of the penicillin treatment the blood culture still showed staph. aureus. The local corneal process gradually cleared up while deep vascularisation indicated an incipient phthisis bulbi.

The main findings after a thorough general examination during the stay of the patient in the hospital were a systolic murmur over the whole region of the heart (endocarditis?) and an increase of the blood sedimentation rate (1h Linzenmeyer), while the blood picture did not show leucocytosis.

Comment

It is fair to assume that the condition under discussion is not as rare as would appear from the scarcity of reports. The not too numerous cases of posterior ring abscess of the cornea published in the literature roughly belong to two groups from the standpoint of aetiology, the traumatic and the metastatic. There is, however, no essential pathogenic difference between these two groups, of which the traumatic comprises the majority. The difference consists in the fact that in cases of metastatic origin the infective agent (deriving from a known or hidden focus) is brought into the inner eye by the vascular route, while in cases of traumatic origin it is introduced into the interior of the eye through the perforating wound. Once established intra-ocularly the microbes most probably act in a similar manner.

Severe acute pyogenic inflammations of the inner eye are regularly accompanied by a more or less pronounced lesion of the corneal endothelium and Descemet's membrane. This condition, frequently manifesting itself clinically in "wrinkling" of the latter membrane, brings about a breakdown of the posterior corneal barrier. Thus infective-toxic substances present in the anterior chamber will be able to enter more or less freely into the posterior lamellae of the cornea. Having entered the corneal tissue these

toxins of bacterial origin may affect the whole posterior part of the cornea and finally the whole cornea, when there is no defensive power left which is the rule in severe cases of this kind. In other cases, however, where defensive forces are mobilized early enough (through the deep ciliary network surrounding the posterior lamellae) the damage to the cornea (varying in degree up to necrosis) may remain confined to its centre. A defensive infiltration is initiated around the damaged cornea. The corneal periphery owing to its better nutrition remains clear and the infiltration gathers around the defenceless centre. This relatively rare condition appears clinically as a posterior ring abscess of the cornea.

Instead of a review of the literature (*a*) the data contained in the table of Gifford & Hunt (1929) are supplemented here, by adding those cases falling within the period of their report and not mentioned by them, and (*b*) comprehensive data from 1929 up to date are compiled, with the exception of a small number of cases to which the present writers had no access. If the latter are not taken into account, thus a total of 42 cases are reported on in the literature, 18 of them belonging to the metastatic and 24 to the traumatic group.

To the columns appearing in Gifford and Hunt's table another one, namely " ocular tension " was added (see Table I) as the factor of increased tension may play a not inconsiderable part in the formation of a posterior ring abscess. Increased tension was mentioned in six out of 18 cases reviewed here, in only one normal tension was expressly stated, while no reference at all was made with regard to ocular tension in the remaining eleven cases.

In the literature dealing with the corneal lesion under discussion two main possibilities were considered which, in a mechanical way, might bring about a rise of tension in severe intra-ocular infection: (*a*) Swelling by massive inflammatory exudation into the vitreous (Fuchs, 1903) and (*b*) blocking of the network of the ligamentum pectinatum by bacteria (Stoelting, 1913) or by a sudden choroidal haemorrhage behind the angle of the anterior chamber (Seale, 1931). Besides the mechanical factors, however, increase of intra-ocular tension is certainly also due to vasomotor changes as well as to disturbances of the osmotic equilibrium: intra-ocular exudate rich in protein will effect retention of fluid and, in consequence, cause rise of tension.

In a state of increased intra-ocular tension the penetration of toxic substances into the posterior part of the cornea, as previously mentioned, will be greatly enhanced the pressure leading to a further diminution of the vitality of the corneal tissue. This damaging effect in conjunction with the main factor of the defensive annular infiltration arriving in time and in sufficient strength, may help in the formation of a posterior corneal ring abscess.

TABLE I

Data from the Literature

Name	No. of cases	Causative factor	Observation and bacteriology	Ocular tension	Result
Herrenheiser (1893)	1	Puerperal septicaemia	—	?	—
*Wintersteiner (1903)	1	Puerperal septicaemia	—	?	—
Stoewer (1907)	1	Injury	Culture from corneal ulcer and from aqueous humour; bacilli of the family of bac. proteus	Increased	Enucleation
Mizuo (1910)	1	Bubonic plague	No bacteriologic work reported	?	Perforation of cornea
—	1	Bubonic plague	No bacteriologic work reported	?	Death
—	1	Bubonic plague	In the suprachoroidal space some bacilli similar to involution forms of plague bacilli	Increased	Enucleation
—	1	Bubonic plague	Plague bacilli mainly in aqueous humour	?	Enucleation death
King (1925) ...	1	?	Smear and culture of secretion pneumococci	Increased	Evisceration
Gifford and Hunt (1929)	1	Injury	Smears and cultures from cut surface of cornea and vitreous. Bac. subtilis	?	Evisceration
—	1	Injury	Smears and cultures from the cornea and vitreous. Bac. subtilis	?	Evisceration
—	1	Injury	Smears and cultures from cornea and vitreous probably Bac. subtilis	?	Evisceration
—	1	Injury	Bacteriologic work not done. In 2 rabbits successful inoculation of vitreous	?	Evisceration
Seale (1931) ...	1	Diabetes, carbuncles	Bacteriologic work not reported	Increased	Death
Barkan (1933)	1	Agranulocytic angina	Blood culture negative	Normal (in the late stage)	Perforation of cornea

* This case is included in Gifford and Hunt's table among Fuchs' ten cases

TABLE I—*continued*

Data from the Literature

Name	No. of cases	Causative factor	Observation and bacteriology	Ocular tension	Result
Herrmann (1934)	1	After exacerbation of trachoma and pannus, small corneal ulcer	Bacteriologic finding negative	?	Remaining 1/10 of vision
Grancini (1937)	1	Streptococcal septicaemia	Streptococci in blood	?	*Perforation* of cornea, death
Tooker (1939)	1	Staphylococcal septicaemia	Staphylococci in blood	Increased	Phthisis *bulbi*
Feigenbaum & Kornblueth (1946)	1	Behcet's disease	Staphylococci in blood and aqueous humour	Increased	Phthisis *bulbi*

Summary

A case of posterior ring abscess of the cornea is described for the first time in Behcet's disease, a chronic septic condition caused by staphylococcus aureus in a constitutionally predisposed individual.

The rôle played by increased intra-ocular tension is stressed which, along with other known aetiological factors, may produce a posterior ring abscess of the cornea.

The cases reported on in the literature are compiled in a table, which is a supplementation of Gifford and Hunt's table published in 1929 and its continuation up to date.

REFERENCES

BARKAN, H.—*Amer. Jl. Ophthal.*, Vol. XVI, pp. 406-408, 1933.

FEIGENBAUM, A. and KORNBLUETH, W.—*Acta Medica Orientat.*, Vol. V, pp. 139-151, 1946.

FUCHS, E.—*Graefe's Arch. f. Ophthal.*, Vol. LVI, pp. 1-31, 1903.

GIFFORD, S. R. and HUNT, C. E.—*Arch. Ophthal.*, Vol. I, pp. 494-500, 1929.

GRANCINI, E.—*Boll. d'Ocul.*, Vol. XVI, pp. 742-752, 1937.

HERRENHEISER, J.—*Prager Zeitschr. f. Heilk.*, Vol. XIV, 1893.

HERRMANN, P.—*Arch. d'Ophtal.*, Vol. LI, p. 671, 1934.

KING, C. L.—*Amer. Jl. Ophthal.*, Vol. VIII, pp. 723-724, 1925.

MIZUO, G.—*Arch. f. Augenheilk.*, Vol. LXV, pp. 1-41, 1910.

SEALE, E. A.—*Brit. Jl. Ophthal.*, Vol. XV, pp. 514-515, 1931.

STOELTING, B.—*Klin. Monatsbl. f. Augenheilk.*, Vol. LI, pp. 5-19, 1913.

STOEWER, P.—*Klin. Monatsbl. f. Augenheilk.*, Vol. XLV, pp. 372-374, 1907.

TOOKER, C. W.—*Amer. Jl. Ophthal.*, Vol. XXII, pp. 526-535, 1939.

MYOPIA AND PSEUDO-MYOPIA*

BY

J. P. SPENCER WALKER

HOVE

VARIOUS theories have been made as to how myopia may progress. I myself have suggested that progression may coincide with an abnormal rate of growth in the adolescent child. My friend, Dr. Dunstan produced statistics which showed that progressive myopia tended to be found in tall, fair, slender children. The weight does not increase proportionately to the increase in height. I still consider that this is one aspect of the problem, but I have always held that there are other causes, for all progressive myopes are not tall, fair and slender. We all have had cases in which the myopia increased but the rate of growth was slow. I now suggest that spasm of accommodation may be an important factor. Very little seems to have been written about accommodative spasm, and, in fact, some authorities consider the condition rare. But, it would appear from cases which I have seen, that it may be commoner than has been supposed, and the fact that it has been found in myopes may point to another cause of progression. We are all familiar with the case which accepts too high a correction, say one or even two dioptres more than the lens indicated by the refraction. It is very tempting to give a patient this added correction, but are we not producing an artificial increase in the myopia? Pseudo-myopia is the same thing as spasm of accommodation, but the term, perhaps, emphasizes a different aspect of the condition. Spasm of accommodation may be found in myopes, emmetropes and hypermetropes, in the young and in the middle-aged. I have used the term pseudo-myopia, as for the moment, I am more concerned with the problem of progressive myopia, but the cases which I am going to quote will include spasm of accommodation in middle-aged as well as adolescent and hypermetropes. My present ideas began to take shape in August, 1944, when a boy was brought to me because he had been rejected for entry to Dartmouth on the grounds that he was myopic, and his mother did not agree with the diagnosis. For once mother was right.

(1) This boy, aged 13½ years, had been examined, in the usual way, for entry to Dartmouth, and had been rejected for "myopia and myopic astigmatism." He was refracted without a mydriatic, but when his mother appealed, he was re-examined after "drops" had been instilled once, and I presume that these drops were homatropine. He was again rejected for the same

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reason. He was then brought to me, when I found that his vision was R. and L.E. 6/6 partly (without glasses), and his refraction without a mydriatic was then R. and L. −0·50 D.Cyl. 180, and his vision with this correction was R. and L. 6/6. His vision was, however, very variable, sometimes being 6/6, and sometimes 6/9 without any lens. After six orthoptic treatments his refraction *under atropine*, administered twice a day for three days, was R. and L.E. *plus* 0·25 D.Sph. *plus* 0·25 D.Cyl. vertical. Obviously, we may call this pseudo-myopia, and the correct diagnosis was of such importance to this boy, that I determined to examine all patients under sixteen under atropine.

(2) A girl who was brought to me in February, 1945, because she refused to wear the glasses, which had been prescribed by someone else, saying that they were uncomfortable, is a typical case. Her age was 12 years, and her vision R.E. 6/24 L.E. 6/36 partly, and she had been given R. and L. −1·25 D.Sph. as glasses for constant use. These she found uncomfortable. Under atropine given twice daily for three days refraction showed her to be *emmetropic*. While under atropine the vision of the right eye was still 6/24, but that of the left eye had risen from 6/36 to 6/24, even though the atropine had been used for three days only. The synoptophore readings were as follows:—Sim: Per: 0, Fusion obtained at 0, Adduction 5, Abduction 5, Rod eso 1, Wing 0-2 exo and Liv: Bin: G: 6 cms. There was intense macular suppression on all tests. She made good progress with orthoptic training. This was a case of emmetropia, which needed such treatment.

(3) A boy, aged 17 years, gave a history of having been examined two years before for "short-sight." The first oculist said he did not need glasses, but the second prescribed R. and L. −0·25 D.Sph. −0·25 D.Cyl. vertical, for constant use. On neither occasion had he been refracted under a mydriatic. His refraction under atropine was R. and L. *plus* 0·75 D.Sph. His vision was R.E. 6/9, L.E. 6/6, but was variable. He was examined on the synoptophore, and the report was:—Sim: Per: plus 1, Fusion obtained at 0, Adduction 20, Abduction 4, Stereopsis present, Wing 3-5 eso, Liv: Bin: G: (objective) 17 cms. "There is marked suppression present with poor convergence and adduction. There does not seem to be any blurring on the synoptophore, which might suggest spasm, but all tests are a great effort, and cause discomfort." In a very few cases spasm may be noted on the synoptophore, but in most cases it is only disclosed by the use of atropine, which should always be used.

(4) A similar condition will be found in adults, for instance, a woman, aged 34 years, complained of headaches. She had been wearing R. and L. −0·75 D.Sph. for general purposes. Her vision

without glasses was R. and L. 6/9 partly. She was interesting because she had no power of convergence, suppressed much, and had photophobia. I shall refer to this combination of suppression and photophobia again later. Her refraction was emmetropic.

(5) A year ago I was asked to see an extremely interesting patient of 50 years of age. She was seen by her doctor on August 3, 1945, who found that her sight was satisfactory, but two days later her vision had failed. When I saw her on August 8, her vision was 6/60 in either eye. The R. vision with plus 1·75 D.Sph. was 6/36, and the L. vision with plus 1·75 D.Sph. was 6/18 partly. I tested her with the Worth lights with the curious result that she suppressed the red light, and saw three Worth dots with the red glass before the right eye, and three Worth dots with the red glass before the left eye. She had slight conjunctival hyperaemia in the right eye, but otherwise appeared to be normal. She had had a severe attack of alopecia, but was now recovered. Her synoptophore report was as follows:—Sim: Per: 0, Fusion obtained at 0, Adduction 20, Abduction 4, Rod eso 2-4, Wing exo 2-4, and Liv: Bin: G: (objective 16 cms.), (*subjective 10 cms.*). "The loss of vision appears to be due to a severe ciliary spasm which should be relieved by atropine treatment later." I kept her under atropine in spite of her age, and she made an excellent recovery. In the following December her synoptophore report was as follows:—Sim: Per: 0, Fusion obtained at 0, Adduction 30, Abduction 6, Rod ortho: Wing Ortho: Liv: Bin: G: (objective 6 cms.), (*subjective 19 cms.*). "Quite comfortable now." It will be noted that her vision had risen to 6/6 without glasses. I saw her again this summer, ten months after the first visit, when her condition was quite comfortable with full vision, and no return of the symptoms.

Sir John Parsons says . . . "excessive contraction of the ciliary muscle; a condition which we call 'spasm of accommodation,' is probably diagnosed more often than it occurs, and that it is only found in young patients." But, however, I have quoted to you one patient of 34 years and another of 50 years.

So far we have only considered cases of pseudo-myopia, but let us consider myopes with pseudo-myopia. But before considering such cases, it is necessary to decide if it is permissable or safe to give orthoptic training to myopes. To the best of my belief it was held that it was dangerous to do so, but I may be mistaken. I was, however, confronted with a case where orthoptic training was much needed, although the patient was a fairly high myope. I hesitated to prescribe this treatment, but after keeping him under observation for a time, I decided that it must be given. He has responded well, and has sustained this response without any increase in the myopia. He was a case of myopia with diplopia and suppression.

(6) He was 14 years of age when he was first brought to me, and was already wearing glasses for myopia. His myopia had increased since the time when these glasses were prescribed, and so I ordered fresh glasses, namely, R.E. -4·25 D.Sph., L.E. -5·0 D.Sph., and with these he saw R.E. 6/6, L.E. 6/18. Six months later his right eye was unchanged, but there was an increase in the myopia in the left, and with the new glasses he saw 6/12 partly with the left eye. He had been bothered with diplopia for some time, in fact that was why he was brought to me. I was doubtful if the myopia in the left eye had increased as much as it appeared, but eventually I was able to prove that the increase was real. His synoptophore report was, however, very informative; it was as follows:—Sim: Per: 0, Fusion obtained at 0, Adduction 5, Abduction 2 (all with glasses), Stereopsis slow, Worth lights 5, Wing 2-4 exo, Liv: Bin: G: (objective test) 7 cms. I was very doubtful if it would be wise to let him have orthoptic training, but in view of the diplopia, which bothered him considerably, I felt justified in letting him try. He responded very quickly, for in a month his adduction, without glasses, was 50, and his stereopsis good. The diplopia was improved, for although he saw 5 Worth dots to begin with, they quickly became 4. The exophoria on the wing had improved to 0-2, and he could converge to 6 cms. What was almost more important, his vision had improved to R. 6/5, L. 6/9 partly with his glasses, and his myopia had not increased.

I will now allude quite briefly to two cases of myopia with pseudo-myopia.

(7) The first was a girl, aged 8¾ years, whose vision was 6/18 when first seen. Under atropine her refraction was R. and L. -0·75 D.Sph. and her vision 6/5 with this correction. There was an increase of 0·50 D.Sph. in the myopia three months later, which remained stationery for a year, but her vision then had fallen to 6/12 with glasses, although the myopia had not increased. The synoptophore report showed she had marked suppression and very poor ductions.

(8) In 1944, I saw a girl, aged 8½ years, whose vision was R. and L. 6/36 partly. Without atropine, her refraction was R. and L. -1·75 D.Sph. and with this correction she was 6/6. Her synoptophore report was as follows:—Sim: Per: plus 1, Fusion obtained at 0, Adduction 8, Abduction 3, Stereopsis present. There was some suppression present but not in any great degree. With orthoptic training, her adduction improved, and her vision came up to 6/12 without glasses, but after six months, her vision fell back to 6/24, but her muscle balance and adduction remained satisfactory. A later report showed the following:—Sim: Per: 0, Fusion obtained at 0, Adduction 20, Abduction 4, Worth lights 4, Rod exo 4, Wing Ortho: Liv: Bin: G: 10 cms. I tested her

again under atropine and found that her refraction was −0.75 D.Sph. I ordered this correction for constant use which put her vision up to 6/6. I consider she is a case of progressive myopia with pseudo-myopia, and it should be noted, that even now, she is not wearing as much correction as she liked on her first visit.

(9) The following case is also of interest. When I first saw her at the end of last year, her vision was 6/60. Her refraction under atropine was R. and L. plus 0·75 D.Sph. and her vision while under the mydriatic was R. 6/36, L. 6/36. Her synoptophore report was as follows :—Sim : Per. 0, Fusion obtained at 0, Adduction 2 blurred, Abduction 7, Worth lights 4, Rod exo 3, Wing 01-2, Liv : Bin : G : (objective 10 cms.), indefinite evidence of ciliary spasm. I kept her under atropine, and she started orthoptic training before the effect of the mydriatic had worn off. Her second synoptophore report was given this summer, and I refracted her again, and found that her refraction was now −0·75 D.Sph. She, therefore, was a case of pseudo-myopia and progressive myopia in a hypermetrope.

It is not always realised that hypermetropes can often have progressive myopia, and I well remember a case of a boy of 13 to 14 years whose refraction was plus 5 D.Sph. He grew extremely rapidly, being about six feet when barely 15 years, and his refraction temporarily became −1. Thus he was in reality a progressive myope of six dioptres. Therefore, the lessening of the hypermetropia in the adolescent is not necessarily the cause for rejoicing.

These cases are of very great interest, and open up an important field for investigation. We all have had cases in which the myopia has not increased, but the vision has fallen, or that the vision is not as high as we might expect. In these cases we have reduced as far as possible the amount of reading and other close work, and in the case of children in schools for myopes, they have been put in the "non-sighted" class. In many, perhaps in a big majority of these cases, the sight has improved with these precautions, but it would be interesting to have a synoptophore report on these children, for I think that in a good many of them we should find that there was spasm of accommodation, suppression or other defects which might be remedied by training. So far, I have not found any evidence that myopia is increased by such training, and I do not see why it should. In any case one would not send a case for treatment unless there was something present in addition to the myopia. I have often found that boys at the school for the partially-sighted which I attend, whose vision with glasses was either about 6/60, or in some cases even less, on leaving school at 16, could see 6/12, or in one or two cases even more, and all that had been done was purely *negative* treatment, namely placing them in the

non-sighted or perhaps partially-sighted class. It is true that up to a point this treatment has had some measure of success, but it is purely negative, and does not remove the child from the partially-sighted category. In my opinion, the partially-sighted person is as great, or even greater, a problem than the blind, for on leaving school how is he to compete with the sighted, even if his vision remains stationary after he has escaped from supervision, or, as is much more likely, he descends into the blind category?

I am not claiming that all these cases are myopes with pseudo-myopia, far from it. Some may be, for it would seem that there are myopes who have a degree of amblyopia, which it should be possible to improve. Let me explain myself. Now why does the vision in these myopes deteriorate, although the myopia is not increasing; and why does the vision improve when reading, etc., is forbidden? Surely, their poor vision is due to suppression or amblyopia. If this proves to be the case, here is an opportunity for *positive* treatment, and the possibility of making them fit to compete with the sighted person on leaving school. I myself, rather like to use these two terms, amblyopia, and suppression for slightly different conditions. I like to use amblyopia for successful suppression, that is suppression without symptoms, and if there are symptoms, then I prefer the term suppression. There are some who appear to be using other terms such as " neglect " instead of suppression. This seems to imply that suppression is a negation, or omission to do something, in this case, to see. But is this really so? Is it not an act, unconscious it is true, but an act, commission if you like, to avoid doing something which is difficult or unpleasant? We none of us like getting our feet wet, but to avoid stepping in a puddle is surely an act, even if it is not consciously done? Then there is the type of suppression, which can be acquired by the refractionist or the bacteriologist. They will use one eye and suppress the other when using their respective instruments. I suppose the term neglect could be applied to this feat, but I am not altogether convinced that they " omit " to see with one of their eyes rather than " suppressing " it.

I have recently had a case which suppressed at a definite distance. This is a new conception to me, that suppression can be acquired at a particular distance, though really there is no reason why this should not be the case, and when I have described the details, I think that you will agree that it supports my suggestion that suppression is an act rather than an omission.

(10) The patient was aged 36 years, and complained of " black-outs " and headaches. She had concussion ten years ago, but had not been subject to headaches since then until quite lately. She was a driver in the A.T.S. Her vision was R. and L. 6/5, and her refraction plus 0·50 D.Sph. She was orthophoric on the

wing test, and could converge to 10 cms. Sim : Per : 0, Fusion obtained at 0, stereopsis was present, but adduction was only 6. She suppressed to a marked degree at "distance," and said that to rest herself after driving she would stare at something close, or would read a book. If she could do this for a sufficiently long time, she would feel refreshed. She responded well to orthoptic training. I have not seen any reference to suppression at a distance, though, when one comes to think of it, there is nothing very strange about it; it is only that the patient suppresses at the place at which his work is normally situated. As the majority of our patients are engaged on near work, for example, typists, clerks or bench-workers, it is only natural that most of them suppress for near objects, but I can well understand that the driver of a car would suppress at a distance.

Earlier I referred to photophobia, and I hasten to admit that I do not altogether understand the connection between suppression and photophobia, and I should like to know if any of you have had similar cases. In one of these cases the photophobia was very marked. When first seen she was a girl of 10 years of age. She had had acidosis when younger, and had had her appendix removed when she was seven. She also had had sinus infection, and her tonsils and adenoids removed. She complained of headaches. Her refraction was plus 1·0 D.Sph. and her vision was 6/6 or rather better. She had poor adduction. After twelve orthoptic treatments she had excellent ductions, was orthophoric and could converge well. Four years later she had marked suppression and very poor adduction. Treatment was again given with the result that there was a very fair improvement. She went back to school, and during the next holidays had further treatment, with further improvement, but summer was approaching, and photophobia began to trouble her. I have always been very reluctant to prescribe a tinted glass, unless there were very good grounds for doing so, but in this case I felt justified, and so ordered Crookes' B.1 glasses and also vitamin A; the latter did not seem to have any effect. She had further treatment in the summer holidays, and by the end of this course she had overcome the suppression for the first time. I am convinced that the suppression would not have been conquered, if the photophobia had not been alleviated, for the real improvement in the suppression started after the prescription of dark glasses. From this and other similar cases, which I have had, it would appear that there is some connection between suppression and photophobia, but I am far from certain what it is. I am inclined to think that the latter is a manifestation of the former.

To sum up these are the chief points :—

(1) All patients under the age of 16, should be refracted under atropine administered twice a day for three days. Especially is

this necessary in those myopes who accept a lens of higher power than their refraction would warrant, or would accept it on a subjective test. In some cases it may be necessary to use atropine for a fortnight, in order to get full relaxation of accommodation.

(2) In pseudo-myopia the vision may be variable.

(3) That investigation of the myopes in a school for the partially-sighted along the lines suggested might be profitable and might throw some light on the nature and treatment of myopia in the adolescent.

(4) That in those cases in which there are symptoms or signs suggesting the need for orthoptic training, the presence of myopia is not necessarily a contra-indication.

(5) That pseudo-myopia is possibly more common than previously supposed, and that it occurs in adults as well as in young people, and also in emmetropes and hypermetropes as well as in myopes.

REFERENCES

DUNSTAN, W. R.—"Visual acuity." *The Medical Officer*, No. 1309, Vol. I, No. 9.

PARSONS, SIR J.—"Developmental myopia and the treatment of myopes." "Diseases of the eye." *Lancet*, October 7, 1933.

SCHURR, C. G.—"Myopia in childhood." *Proceedings Medical-Chir. Society, Brighton*, May 3, 1928.

WOOD, D. J.—"Calcium deficiency in the blood—myopia." *Brit. Jl. Ophthal.*, Vol. XI, No. 5, May 1927.

WALKER, J. P. S.—"Progressive myopia." *Brit. Jl. Ophthal.*, Vol. XVI, No. 8, August, 1932.

THE PROBLEMS OF GLAUCOMA*

BY

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HRADEC KRÁLOVÉ, Č.S.R.

THE causes of glaucoma are still unknown, in spite of the considerable progress in science and satisfactory therapeutic results. Our knowledge today is the result of the work of generations, but in spite of its great value, we are well aware that the right solution is still wanting. The theories were always arrived at correspondingly to the discoveries in the histology of experimental pathology. It is neither advisable nor possible to do justice even in brief to all the opinions which have been expressed about glaucoma or to survey them all.

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It would be a mistake to look upon glaucoma as a single entity, created only by a single and certain cause. The variety of its forms repudiates such a conception.

In accordance with the progress of science we have been forced in the last years to correct some opinions we were accustomed to. The advancing research of the vegetative nervous system, of the mid-brain centre connected with the pituitary gland and the discovery of the nocifensory system has thrown a new light on many problems. Glaucoma has already previously been compared with oedema and I agree that this comparison is justified to a great degree. I therefore think it right to call attention to the present views on oedema and to attempt to find out whether there is a parallel between oedema and glaucoma.

According to its origin we distinguish today inflammatory, cardiac, nephritic oedema and the oedema of Quincke.

One can easily find a parallel between the inflammatory oedema and glaucoma. A typical example of this group is certainly the glaucomatous iritis. Until now it has been nearly exclusively upheld that an increase of the tension arises from the blocking of the drainage system in the anterior chamber of the eye by an exudate. This is to a certain degree probable, but it has already been pointed out previously that it would not be correct to charge only this cause. Thus Schmidt-Rimpler draws attention to the fact that a great number of cases of iritis, sometimes even with a thick exudate, are not accompanied by secondary glaucoma. A good many cases have been described, where the blockage has been proved with the great probability, but on the other hand many have been recorded where this proof has been entirely absent. There is no reason why we should not think that oedema arises in the eye in the same way as anywhere else in the body. Of course, in the other parts of the body oedema is not restricted to such an extent as in the eye where the enlargement of the bulk of the tissue is limited by the relatively inelastic coat of the eyeball. If, however, the tension rises in the capillary blood vessels of the eye, followed by a speedy reduction of oxyhaemoglobin, thus causing defective oxygenation and an accumulation of metabolites, which osmotically attract water, we have also to admit a tendency towards oedema. Since the coat of the eyeball is to a very small extent elastic and therefore incapable of compensation, a greater strain is laid by such a swelling upon the drainage of the aqueous. The regulation of the increased tension by paracentesis, which is supposed to remove the exudate which is the cause of the blockage, is often quoted in support of the opinion that the iridocyclitic glaucoma is caused by blockage of the ducts with exudate (Schieck). At

the same time it is of course possible that paracentesis of the chamber is successful because it removes the excessive amount of metabolites. We are well aware that after the disappearance of the inflammation, i.e., after the disappearance of the pathological amount of metabolites, the glaucoma disappears, as it never outlasts the inflammation, although it would be quite possible to expect the increased pressure to last longer in view of the blocking of the anterior chamber. May I add that according to my impression injections with hypertonic solutions have in such cases a relatively good effect. I have very often tried to apply in such cases not only hypertonic glucose but also hypertonic saline. On the whole I can say that it often stood the test.

Similarly, it is easy to find a parallel to cardiac oedema among diseases with increased tension of the eye, *viz.*, the typical secondary glaucoma which occurs in the case of thrombosis venae centralis retinae. The pathogenesis of this glaucoma is again explained by the blocking of the anterior chamber by the products of decomposition which arise from thrombosis. Elschnig looked upon anterior synechia, which sometimes occurs, as a mechanical and non-inflammatory process, which is caused by the pressure of the swollen lens. If one can compare the glaucoma complicating blocking of the central vein with cardiac oedema, the explanation is simple. The bad circulation produces again metabolites which accumulate in the eye, attract osmotically water and cause intraocular oedema. According to this explanation therapeutic efforts are extremely difficult. It is natural that the main interest of this comparison is directed towards primary glaucoma.

If we want to gain from this comparison with the oedema, we must first of all look for a parallel to the oedema of Quincke. It appears namely that the allergic mechanism, which is influenced by the pituitary gland, mid-brain and the vegetative nervous system may be common to both. Glaucoma has been considered for some time as an alteration of the tone of the sympathetic system. Although it was not possible to account for all the circumstances this opinion has been maintained up to the present day. This opinion was mainly supported by the favourable effect of drugs exciting the para-sympathetic, by which it is possible to diminish the pressure in the eye. Some experiments, however, which invalidate the above mentioned doctrine, do not fit into the pattern. The results of experiments consisting in excitation of the sympathetic varied considerably and those of the treatment of glaucoma by extirpation of the sympathetic were vague. After excitation of the sympathetic, an increased pressure followed for a short period and thereafter a long lasting decrease of pressure.

After some time the pressure reverted, according to the literature, to normal or increased. The opinion that glaucoma cannot be due to tone of the sympathetic system was therefore doubted since through irritation after a transitory increase, a decrease of tension sets in. This apparently vague effect of the increased tone of the sympathetic can be easily explained. All actions of life are regulated by the tension of the sympathetic and para-sympathetic. By these actions oscillations are produced. If an increased tone of the sympathetic arises, the reaction of the para-sympathetic ensues immediately and restores balance of the vegetative system. This regulation, however, is by no means precise, and the para-sympathetic response is excessive. Until balance is regained, the oscillation of both regulating elements goes on. This oscillation can be represented by waves which die away.

The same phenomenon ensues also after subconjunctival application of adrenalin, which is a typical drug exciting the sympathetic. We know that adrenalin is a stimulus for the sympathetic, *i.e.*, a substance which the sympathetic sets free and which has its effects on the organs. In a voluminous literature we can find many statements about the effect of adrenalin on glaucoma. Adrenalin was even used successfully to decrease intra-ocular pressure. This was especially successful with glaucoma simplex. Opinions, however, about the effect of adrenalin upon glaucoma differ considerably. While it is by some recommended, others warn against its use, because it has happened that by application of adrenalin a glaucomatous attack has been produced (Imre, Jess, MacCallan, Parsons, Gamo Pinto, Rubert and others). Here again a considerable incongruity in the effect of this substance ensues. Adrenalin works for a short period only, because of its quick decomposition. It produces an intensive mydriasis which is remarkable for its effect on the sympathetic. Adrenalin produces in addition a transitory vaso-constriction, which soon disappears and is replaced by a vaso-dilation. The effect on the tension of the eye varies again according to the literature. Knapp, for instance, instilled drops of adrenalin in 65 cases of primary glaucoma five times at one minute's interval. In 40 cases the tension remained unchanged, in 20 it was a little lower, and in 5 cases higher. I think that these incongruities in the effect of adrenalin can be easily accounted for. I therefore measured the tension every thirty seconds in 22 cases after subconjunctival injection of adrenalin. In 17 cases I found with certainty a slight but transitory increase of tension, which was immediately followed by a decrease. In one case the tension was increased for a period of three minutes, then it returned to normal. The remaining 4 cases did not show any measurable change in the way of increase, but after about two minutes a

slight decrease of tension ensued. With regard to the above mentioned measurements it is probable, that adrenalin, working only for a short period, increases the tension of the eye at first and thus provokes a reaction of the para-sympathetic, which endeavours to regain the vegetative balance and thus reduces the tension quite considerably.

Such an effect of adrenalin presupposes a normal reaction of the para-sympathetic. This might even be the reason why adrenalin provoked in some cases of inflammatory glaucoma an increased tension. I observed the tension in 6 cases, where the eye was atropinised. After a short increase in tension in 5 cases, it returned to normal. In one case I did not succeed in recording a change. In view of the small number of measured cases I cannot draw any conclusions.

It was formerly objected to the sympathetic theory of glaucoma, that glaucoma is not in any way affected by the cutting of the sympathetic. Roszin proved in 1926-27 that adrenalin has a great effect on the eye even after the cutting of the sympathetic. It is, however, possible to object that it is not probable that the tone of the sympathetic caused by adrenalin is actually the cause for a state which is characterized by such remarkable symptoms. I am going to make use of a recent invention. As I mentioned before, adrenalin causes a transitory vaso-constriction of the blood vessel, which quickly disappears and is replaced by vaso-dilatation. Vaso-constriction can, however, be prolonged by adding thyroxin. The effect of adrenalin can be accentuated by thyroxin. We can therefore assume an accentuation of the tone of the sympathetic owing to various hormonal causes which influence the vegetative centres. It is known today that the effect of adrenalin can be accentuated not only by thyroxin, but we also know that the parahormone and the hormones of the basophilic cells of pituitary glands are especially closely connected with the sympathetic system. Insulin and the assumed hormone of the thymus are especially closely connected with the para-sympathetic system.

A series of articles describes the relation of glaucoma to the thyrotoxicoses. The occurrence of glaucoma in connection with Basedow's illness is no rarity. Hyperpara-thyroses are of course very rare and it is difficult to prove this theoretical conclusion by practical experience. In this connection Csapody's case only can be quoted, who observed an increased tension in connection with the pathological fragility of the bones with blue sclerotics.

In the case of basophilic adenoma or of hyperplasia of the basophilic cells of the anterior lobe hypertension of the eye has been observed (Dérer and Kopf, Tatár, Grosz).

Insulin has the opposite effect. Acardi, Ascher, Kadlicky, Westergaard and others report its effect in lowering the tension.

I have mentioned above that it would be possible to explain cases where glaucoma after the injection of adrenalin was brought about as a result of the non-functioning of the para-sympathetic. This non-functioning of the para-sympathetic is most likely caused, according to our yet incomplete knowledge, through the activity or rather inactivity of the mid-brain. The centres of the mid-brain are specially para-sympathetic. Their damping can be effected by mental shock, by conditioned reflexes, by degenerative diencephaloses or by inflammatory diencephalites.

Also it is possible to look for a causal connection with the glaucoma. We know, how often an attack of acute glaucoma (*i.e.*, a glaucoma where adrenalin by experience cannot be applied) occurs after excitement. It may therefore be assumed that the vegetative imbalance was further increased by the damping of para-sympathetic centres of the midbrain.

The vegetative regulation can never be represented by a straight line, but it can be best shown by a wavy line with a regular rhythm. There is a parallelism between this rhythm and that of day and night. The sympathetic has a prominent katabolic effect and incites the organism to increased activity. The sympathetic is preponderant during the day. Para-sympathetic with acetylcholine, which is its nervous stimulus, controls the anabolic actions, *i.e.*, the processes of restitution. The para-sympathetic reigns at night. In addition to this long wave rhythm there is a small oscillation in a short wave rhythm. The tension in glaucoma simplex is according to our experience and the literature greatest in the morning hours and slightest in the evening. The tension is therefore greatest at the time when the sympathetic starts its work, and smallest when the para-sympathetic begins to work. The night-morning start of the sympathetic is of course counterbalanced by the para-sympathetic which tends to reduce the tension during the day to normal. During the day it works the other way round. The course of the tension between those two points of this recurring rhythm depends on the continuous compensating effect of both constituents, which try to keep a balance. In the aetiology of glaucoma mere mechanical moments play their important part as well, mainly anatomical obstacles of drainage proved by histological examinations.

With all these interconnections I have tried to prove, though incompletely, the parallel between glaucoma and oedema. Apparently it most resembles the oedema of Quincke.

To this attempt to explain glaucoma may I add briefly the therapeutic results, most of which are of course not novel. In a conservative way it is possible to control glaucoma as follows:

1. The restoring of the vegetative imbalance

(*a*) raising the tone of the para-sympathetic (pilocarpine, physostigmine) blocking the choline esters for a short period, the new American DPF (di-isopopylfluorophosphate) working by the same mechanism, but for a longer period and a series of drugs related to acetylcholine.

(*b*) paralysing the sympathetic (this our present pharmacological explanation). Ergotamin has been used and I myself tried to use benzodioxan, but the effect of these drugs was not remarkable, it only increased the effects of miotics.

(*c*) Vegetative imbalance can also be adjusted with shocks. Moretty compensated glaucoma with autohaemotherapy. I myself often lowered the tension of the eye during an attack for 5-24 hours by a subconjunctival injection of the serum of the patient's blood. Stásnik compensated the glaucoma with histamin. These slight successes could justify the opinion that some glaucomas arise on an allergic basis.

2. Removing accentuating causes (I succeeded recently in compensating two glaucomas accompanied at the same time by thyrotoxicosis only, after having administered pilocarpine with vitamin A, which is the antagonizer of thyroxin). It would be probably right to look in all cases of glaucomas for the accentuating cause. Where, however, the accentuating cause is merely anatomical, a conservative therapy would only succeed to a minor degree.

In this article I wished to apply some of our knowledge about oedema to the problems of glaucoma. I am well aware that many assumptions without much proof have been expressed. It appeared to me, however, that from the point of view of these new opinions many problems are identical or overlapping.

REFERENCES

BRÜCKNER.—*Ophthalmologica*, Vol. CVI, p. 200.

HEILMEYER.—*Lehrbuch d. spez. path. Physiol.*, p. 428, 1944.

MARX.—*Der Wasserhaushalt*, p. 178, 1935.

PETERS.—*Das Glaukom.*

SHEN, J.—*Physiology*, p. 51, 1937.

TATÁR.—*Graefe Arch. f. Ophthal.*, p. 253, 1946.

Traité de chimie organique, Vol. XII, p. 630.

AN INVESTIGATION OF "NORMAL" ON THE SYNOPTOPHORE*

BY

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FOR many years the synoptophore has been used as a means for measuring and treating ocular muscle imbalance. No work has apparently been done to show whether the results are repeatable, whether the range of normal is wide or narrow, or whether there is a high correlation between the synoptophore and other methods of measuring ocular muscle imbalance.

In this investigation 98 young adult males (aircrew) and 2 W.A.A.F., aged 18-30 years, were examined to determine:—

(*a*) The "normal" range of synoptophore measurements.
(*b*) The repeatability of the measurements.
(*c*) The results using two different methods of measurement.
(*d*) A comparison between the synoptophore simultaneous perception readings and the commonly used Maddox rod test for heterophoria.

None of these young men and women complained of any symptoms referable to their eyes.

Apparatus

1. *Synoptophore* with scale in prism dioptres. The calibration of the instrument remained constant throughout the experiment.

Facilities to check the zero position on the scale were not available, but there was no reason to doubt that the zero was reasonably accurate.

2. *Red Maddox rod* and separate prisms, with spotlight at 20 feet.
3. *Millimetre rule.*
4. *Snellen test type at 20 feet.*

Methods

Each subject had the following measurements done:—

(*a*) *Visual Acuity.* No refractions were done, and no corrections were worn for synoptophore measurements.

(*b*) *Maddox rod.* The Maddox rod was placed before the right eye, and prisms were used to measure the heterophoria. This procedure was then carried out with the rod before the left eye. In a few cases a slight difference between the two readings was found. The

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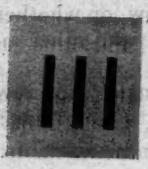
difference was rarely as much as 1 prism dioptre. Since synoptophore measurements were taken with the right eye fixing, in the analysis of results that follows Maddox rod readings with the rod before the left eye (right eye fixing spot light) have been used.

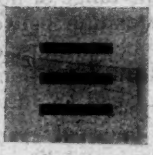
(*c*) *Interpupillary distance.* This was measured from centre of the right pupil to the centre of the left pupil.

(*d*) *Synoptophore.* This was adjusted for the interpupillary distance of each subject, and readings taken as follows:—

1. *Simultaneous perception.* Slides 1 and 2 (Fig. 1) were used, slide 1 before the right eye and slide 2 before the left.



SLIDE 1



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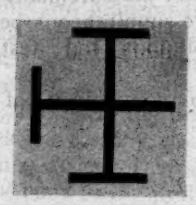

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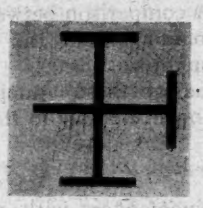

SLIDE 4

FIG. 1.

Method A. The right synoptophore tube was set at 0, and the subject fixed the centre of slide 1 with his right eye. The left synoptophore tube with slide 2 was set at approximately −15 prism dioptres. The subject was asked to move the left synoptophore tube until the two sets of lines appeared superimposed. The synoptophore scale reading was recorded. This measurement was made four times.

Method B. The above was repeated, but with the left synoptophore tube set at approximately +15 prism dioptres.

2. *Fusion.* Slide 3 for the right eye and slide 4 for the left eye were used (Fig. I).

Method A. This was exactly as for method A above. The measurement was made twice.

Method B. The above was repeated, but with the left synoptophore tube set at approximately +15 prism dioptres.

3. *Abduction.* Slides 3 and 4 were again used. Fusion was first

obtained using method A above. The synoptophore tubes were then locked, and the subject instructed to look at the centre of the picture. Abduction was measured by slowly turning the abduction screw until the pictures no longer appeared fused. The reading was noted, and the measurement repeated.

4. *Adduction.* Fusion was once more obtained by method A, using slides 3 and 4. The synoptophore tubes were locked, and the subject instructed to look at the centre of the picture. He was told that the picture would probably get blurred, and that if it did, he was not to try to see it clearly. He was also told that the picture would gradually appear to get smaller. Adduction was measured by slowly turning the adduction screw until the pictures no longer appeared fused. The reading was noted and the whole procedure repeated.

Results

I. *Visual acuity.* This varied between 6/5 and 6/18. There was only one person with visual acuity as low as 6/18.

II. *Synoptophore.* All readings are in prism dioptres.

(i) *Simultaneous perception.*

Method A	1	2	3	4
Mean	—2·84	—3·08	—3·15	—3·16
Standard deviation	2·24	2·33	2·15	2·05

The means of readings 1 and 4 show the greatest difference. The standard error of the difference between these two means is 0·304. The difference between these means is not significant, being only the same as the standard error.

Method B	1	2	3	4
Mean	—1·68	—1·48	—1·22	—1·31
Standard deviation	2·28	2·15	2·29	2·29

The means of readings 1 and 3 show the greatest difference. The standard error of the difference between these two means is 0·313, which again is practically the same as the difference. No significant difference therefore occurs between the means.

Joint consideration of methods A and B gives the following series of standard errors:—

1st reading of both methods (means —2·84 and —1·68) S.E. =0·320
2nd reading of both methods (means —3·08 and —1·48) S.E. =0·317
3rd reading of both methods (means —3·15 and —1·22) S.E. =0·314
4th reading of both methods (means —3·16 and —1·31) S.E. =0·307

The differences between the means are all significant, as they vary from 3 to 6 times the respective standard errors.

The test-retest correlation of the first and second readings using method A is +0·926.

(ii) *Fusion.*

Method A	1	3
Mean	—3·12	—3·04
Standard deviation	2·04	2·28
Method B		
Mean	—1·82	—1·49
Standard deviation	2·23	2·27

The standard error of the difference between the means of the first and second readings of method A is 0·306.

The standard error of the difference between the means of the first and second readings of method B is 0·318.

The differences between the means are not significant, as in both cases the differences are less than the respective standard errors.

The standard errors of the difference between the means of the fusion readings obtained by method A and by method B, are as follows:—

1st reading of both methods (means —3·12 and —1·82) S.E. =0·302
2nd reading of both methods (means —3·04 and —1·49) S.E. =0·312

The differences between these means are significant, as they vary from four to five times the respective standard errors.

The standard errors of the difference between the means of the simultaneous perception readings and the fusion readings, are as follows:—

1st reading of S.P. method A and of fusion method A
(means —2·84 and —3·12) S.E =0·304
2nd reading of S.P. method A and of fusion method A
(means —3·08 and —3·04) S.E. =0·325
1st reading of S.P. method B and of fusion method B
(means —1·68 and —1·82) S.E. =0·319
2nd reading of S.P. method B and of fusion method B
(means —1·48 and —1·49) S.E. =0·313

The differences between these means are not significant, since each difference is less than the corresponding standard error.

(iii) *Abduction*	1	2
Mean	—7·61	—6·91
Standard deviation	2·19	2·57

The standard error of the difference between the means is 0·34. The difference between the means is therefore on the borderline of significance, as it is equal to twice the standard error.

(iv) *Adduction*	1	2
Mean	+27·09	+29·83
Standard deviation	22·93	22·05

The standard error of the difference between the means is 3·18. The difference between the means is less than the standard error, and therefore is not significant.

III. *Maddox rod.*

Mean	+0·89
Standard deviation	1·18

The correlation co-efficient of the first simultaneous perception readings (method A) and the Maddox rod readings is +0·479.

Conclusions

The following conclusions apply to those examined:—

1. *Synoptophore.*

(i) *Simultaneous perception*—(*a*) Analysis of the results shows that there is no significant difference between four readings taken in succession on the same person by the same method (A or B).

(*b*) There is a significant difference between readings taken by method A and method B. The difference between the means obtained by method A and method B are shown below for four successive sets of readings:—

1	2	3	4
1·16	1·60	1·93	1·85

(*c*) The readings obtained by method A are more exophoric than those obtained by method B.

(*d*) The test-retest correlation of the first and second simultaneous perception readings (method A in each case) is very high, +0·926.

(ii) *Fusion*—(*a*) There is no significant difference between two readings taken in succession on the same person by the same method (A or B).

(*b*) There is a significant difference between readings taken by method A and method B. The difference between the means obtained by method A and method B are shown below for two successive sets of readings:—

1	2
1·30	1·55

(iii) *Simultaneous perception and fusion*—Readings obtained by means of simultaneous perception slides do not differ significantly from those obtained by the use of fusion slides, provided the slides are used in the same manner (*e.g.* method A or method B).

(iv) *Abduction and Adduction*—No significant difference was found between two successive readings in the case of either abduction or adduction.

2. *Maddox rod and synoptophore.*

(i) Correlation co-efficient = +0·479.

There is some correlation between the Maddox rod and synoptophore readings. It would appear that the two devices do not measure identical qualities of heterophoria.

(ii) The results obtained by the synoptophore are more exophoric than those obtained by the Maddox rod. (See Fig. 2).

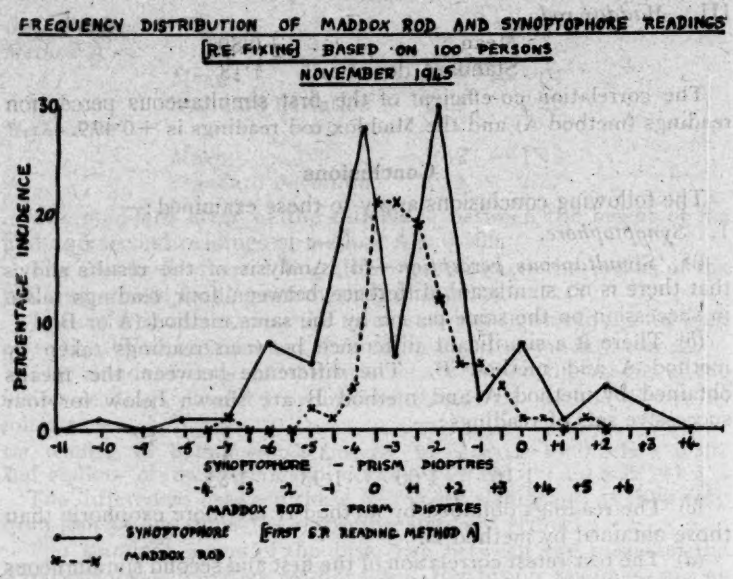
FREQUENCY DISTRIBUTION OF MADDOX ROD AND SYNOPTOPHORE READINGS
(R.E. FIXING) BASED ON 100 PERSONS
NOVEMBER 1945
PERCENTAGE INCIDENCE
30
20
10
0
-11 -10 -9 -8 -7 -6 -5 -4 -3 -2 -1 0 +1 +2 +3 +4
SYNOPTOPHORE - PRISM DIOPTRES
-4 -3 -2 -1 0 +1 +2 +3 +4 +5 +6
MADDOX ROD - PRISM DIOPTRES
SYNOPTOPHORE (FIRST S.P. READING METHOD A)
MADDOX ROD

FIG. 2.

(iii) The synoptophore readings are spread over a wider range than the Maddox rod readings for the same people. (See Fig. 2).

Discussion

With both the simultaneous perception and the fusion slides there was a statistically significant difference between the results obtained with method A as compared with method B. The difference was in the same direction for both simultaneous perception and fusion. In general, the reading with method B was more esophoric than with method A. The difference could be explained in part at least by the convergent initial position of the slides using method B. Further, fusion and simultaneous perception are not in practice, point features, but rather events which occur within a certain range. The response given by the individual would therefore be dependent on whether he started from the convergent or divergent position. In starting from the convergent position he would enter first the more esophoric portion of the range of fusion, and similarly from the divergent position when the more exophoric portion would be first entered. This shows that from the practical point of view it is essential:—

(*a*) To make the measurements on each occasion with the tube in the same initial position.

(*b*) To employ always the reading from the convergent position or the reading from the divergent position, but not sometimes from

the one and sometimes from the other. For some work the mean of the readings from the two opposite initial positions could be used.

It is obvious from this group of symptom-free people that there is a wide range of normality. If the range "mean — standard deviation to mean + standard deviation" is taken as normal, then the following are suggested as the figures for the normals of the various measurements:—

Simultaneous perception and fusion.

	Method A	—0·5 to —5·5 prism dioptres
	Method B	+1·0 to —4·0 prism dioptres
Abduction	... ...	—4·5 to —10 prism dioptres
Adduction	... ...	+4 to +52 prism dioptres

Recommendations

It is recommended that subjective measurements on the synoptophore be taken by either method A or else by method B, as the results obtained by method A differ from those obtained by method B. Of the two methods, it is suggested that method A is preferable to method B.

Summary

Ninety-eight aircrew personnel and two W.A.A.F. were examined on the synoptophore and Maddox rod. In addition each person had his visual acuity and inter-pupillary distance measured.

On the synoptophore, simultaneous perception and fusion were both measured subjectively by two different methods (A and B). Four simultaneous perception readings and two fusion readings were taken by each method. It was found that there was no significant difference between successive simultaneous perception and fusion readings obtained by the same method (either A or B), but there was a significant difference between simultaneous perception and fusion readings obtained by method A as compared with method B. The test-retest correlation for simultaneous perception was high (+0·926), and the figures for fusion suggest that the test-retest correlation there is also high.

Two abduction and two adduction measurements were taken in succession on each person on the synoptophore. No significant difference was found in the two successive readings of either abduction or adduction.

By studying the means and standard deviations of the various synoptophore readings, a "normal" range was calculated for each type of reading. The range of "normal" adduction readings was found to be very large.

Maddox rod and synoptophore readings (right eye fixing in each case) were compared, and the correlation co-efficient was found to be +0·479. The synoptophore readings were more exophoric and spread over a wider range than the Maddox rod readings.

APPENDIX

Subject No.	V. A. R.	V. A. L.	M. R.	P.D. mm.	Synoptophore: Simultaneous Perception Method A 1	Method A 2	Method A 3	Method A 4	Method B 1	Method B 2	Method B 3	Method B 4	Fusion Method A 1	Method A 2	Method B 1	Method B 2	Abduction 1	Abduction 2	Adduction 1	Adduction 2	Subject No.
1	6/9	6/5	+1	65	−4	−4	−4	−4	−2	−2	−2	−2	−2	−2	−2	−2	−8	−6	+44	+46	54
2	6/5	6/5	+1	69	+2	0	0	0	+1	+2	+2	+2	−4	−4	+6	+4	−4	−4	+50	+34	55
3	6/5	6/5	−1½	68	−4	−4	−3	−3	0	−2	−2	−2	−4	−4	−2	−2	−9	−10	+13	+14	56
4	6/6	6/6	−½	64	−6	−6	−8	−7	−5	−4	−5	−4	−4	−5	0	+2	−6	−6	+50	+68	57
5	6/6	6/6	0	63	0	−1	−2	−2	−2	−1	−2	−2	−5	−5	−4	−5	−9	−10	+15	+12	58
6	6/5	6/5	0	70	−1	−3	−2	−3	+1	+1	+2	+1	−2	−4	−2	−3	−9	−8	+56	+50	59
7	6/5	6/6	+1½	60	+2	+2	+2	+2	+5	+5	+6	+5	0	0	+3	+4	−4	−3	+60	+56	60
8	6/6	6/6	0	63	−2	−4	−3	−4	−4	−3	−2	−4	−2	−2	−2	−3	−7	−5	+16	+16	61
9	6/9	6/5	0	67	0	−6	−4	−6	−4	−2	−2	−2	−4	−4	−4	0	−7	−6	+22	+18	62
10	6/6	6/6	+½	64	+2	+2	+2	+2	+4	+2	+2	+2	0	−1	−1	−1	−4	−2	+53	+50	63
11	6/6	6/6	+1	65	+1	+1	0	+1	0	0	+1	+1	+1	+4	0	−1	−9	−8	+26	+18	64
12	6/5	6/5	0	64	+3	+2	+3	+3	+3	+3	+3	+3	+1	0	−1	−1	−6	−4	+2	0	65
13	6/5	6/5	+1	61	−4	−6	−4	−4	−2	0	−1	−2	−4	−5	−3	−2	−9	−9	+70	+106	66
14	6/6	6/6	0	65	−2	−3	−3	−2	0	0	+1	+1	−3	−5	−4	−2	−12	−10	+22	+24	67
15	6/6	6/5	0	66	−5	−6	−6	−6	−5	−4	−5	−5	−6	−5	−5	−5	−9	−9	+16	+16	68
16	6/6	6/6	+2	67	−5	−5	−4	−4	−3	−4	−3	−3	−4	−4	−3	−2	−9	−8	+18	+12	69
17	6/6	6/6	+3	65	−1	0	0	0	0	−2	−1	−1	0	0	0	0	−8	−9	+10	+18	70
18	6/5	6/5	+1	63	0	0	0	0	0	0	+2	+2	−2	−3	0	0	−5	−4	+20	+22	71
19	6/9	6/6	+½	69	−5	−4	−4	−4	−4	−4	−4	−4	−4	−4	−4	−2	−10	−8	+14	+14	72
20	6/9	6/6	+5	69	−4	−3	−3	−4	−2	−2	−2	−1	−2	−2	−1	−2	−5	−4	0	0	73
21	6/12	6/9	+2	67	−7	−7	−8	−10	−4	−4	0	0	−6	−8	−4	−4	−12	−12	+12	+8	74
22	6/6	6/6	+½	67	−4	−5	−5	−5	−4	−4	−4	−4	−3	−4	−2	−3	−8	−7	+13	+14	75
23	6/5	6/18	0	66	−4	−2	−4	−3	−3	−2	−3	−2	−3	−4	−2	−2	−6	−6	+22	+20	76
24	6/5	6/6	+3½	64	+2	+2	+2	+3	+4	+4	+4	+4	+4	+4	+5	+6	−4	−4	+22	+17	77
25	6/9	6/9	+3	63	−2	−2	−2	−2	+2	0	0	0	−2	−4	0	+2	−8	−6	+14	+12	78
26	6/6	6/6	0	64	−5	−5	−5	−6	−5	−4	−4	−4	−4	−4	−2	−2	−9	−10	+12	+43	79
27	6/6	6/6	0	64	−4	−4	−4	−4	−3	−2	−2	−3	−5	−6	−4	−2	−10	−8	+9	+14	*80
28	6/12	6/12	−½	64	−2	−4	−4	−5	−4	−2	0	0	−2	−4	−2	−1	−10	−12	+50	+37	81
29	6/6	6/6	0	64	−4	−4	−4	−4	−4	−4	−3	−4	−5	−6	−6	−5	−12	−10	+8	+46	82
30	6/6	6/6	+2	66	−2	−2	−2	−2	−2	0	−1	−1	−2	−2	−2	−2	−7	−5	+12	+34	83
31	6/6	6/6	0	67	−2	−2	−3	−3	−2	−2	−2	−2	−5	−5	−2	−1	−10	−10	+18	+24	84
32	6/9	6/6	+1	63	−1	−1	−1	−1	−1	−1	0	−1	+1	+1	0	0	−4	−4	+18	+14	85
33	6/5	6/5	0	60	−4	−2	−2	−3	−3	−2	−2	−2	−3	−3	0	0	−11	−12	+6	+4	86
34	6/5	6/5	+1	66	−4	−5	−5	−5	−2	−2	−2	0	−4	−4	−4	−2	−9	−8	+15	+12	87
35	6/5	6/6	0	65	−4	−4	−4	−4	−3	−3	−2	−2	−6	−6	−5	−4	−10	−8	+18	+26	88
36	6/5	6/5	+½	65	−4	−2	−3	−4	−4	−2	−2	−1	−2	0	0	+2	−4	−6	+20	+20	89
37	6/9	6/6	+½	61	−2	−4	−4	−2	+2	+2	+2	+1	−5	−7	−1	0	−9	−8	+20	+15	*90
38	6/5	6/6	+2½	64	−4	−4	−2	−4	−2	−2	−4	−2	−4	−5	−3	−2	−6	−6	+38	+40	91
39	6/6	6/5	+1	63	−2	−2	−2	−2	−2	−2	−2	−2	0	−1	−1	−1	−4	−2	+38	+44	92
40	6/5	6/5	+½	65	−2	−2	−4	−4	0	−2	−2	−2	−4	−3	+1	+1	−8	−8	+32	+33	93
41	6/9	6/9	0	67	−2	−2	−2	−2	+2	0	0	0	+3	−4	−2	−2	−5	−5	+10	+13	94
42	6/5	6/5	+2½	70	−2	−1	−1	0	+2	+2	+2	+2	0	+1	−1	0	−6	−6	+78	+80	95
43	6/6	6/6	0	70	−5	−4	−5	−4	−2	−1	−2	−1	−7	−6	−6	−4	−8	−9	+10	+8	96
44	6/6	6/6	+½	62	0	−2	−3	−2	0	−1	−1	0	−3	−2	0	0	−7	−4	+10	+2	97
45	6/6	6/6	+½	66	−4	−4	−4	−4	−2	−2	−1	−2	−3	−4	−2	−2	−7	−6	+6	+2	98
46	6/5	6/5	+1	67	−3	−3	−3	−4	−2	−2	−2	−1	−4	−4	−2	−1	−6	−5	+34	+40	99
47	6/6	6/9	+1½	63	−6	−6	−4	−5	−2	0	−2	−2	−6	−5	−4	−2	−8	−8	+14	+20	100
48	6/6	6/6	+1½	59	−8	−9	−8	−7	−4	−3	−3	−2	−6	−6	−2	+1	−10	−8	+90	+60	
49	6/9	6/6	+1½	62	−4	−2	−4	−3	−2	−2	−2	−2	−2	−3	−2	−2	−6	−6	+16	+18	
50	6/6	6/5	+1½	62	−2	−2	−4	−4	−3	−3	−2	−2	−4	−4	−2	−2	−8	−8	+10	+8	
51	6/5	6/5	+½	68	−5	−5	−4	−5	−4	−4	−3	−2	−5	−4	−5	−5	−8	−8	+8	+4	
52	6/6	6/6	+2½	64	−4	−5	−5	−5	−4	−2	−4	−5	−2	−2	−2	−2	−10	−9	+8	+8	
53	6/6	6/6	+½	63	−2	−4	−4	−4	−3	−3	−2	0	−3	−2	−3	−2	−8	−6	+90	+50	

*W.A
Made

APPENDIX—*continued*

Subject No.	V.A. R.	V.A. L.	M. R.	P.D. mm.	Synoptophore: Simultaneous Perception Method A 1	2	3	4	Method B 1	2	3	4	Fusion Method A 1	2	Method B 1	2	Abduction 1	2	Adduction 1	2
54	6/5	6/5	+½	66	−6	−6	−6	−6	−5	−5	−4	−6	−7	−6	−6	−6	−10	−8	+120	+102
55	6/5	6/5	+1½	66	−3	−3	−4	−4	−2	−2	0	−4	−4	−4	0	0	−7	−5	+38	+34
56	6/9	6/6	+½	64	−2	−3	−4	−4	−2	−2	−1	−2	−2	−3	0	−1	−8	−7	+28	+28
57	6/5	6/5	0	65	−2	−4	−4	−4	−3	−3	−4	−3	−2	−3	−4	−3	−6	−6	+28	+58
58	6/9	6/9	+1½	60	−2	0	−2	−2	−2	−2	0	−1	0	0	+1	0	−4	−2	+32	+28
59	6/12	6/6	+½	69	−2	−2	−2	−3	−2	0	+2	0	−2	−2	−1	0	−12	−14	+34	+38
60	6/6	6/6	+1	61	−4	−4	−4	−4	−3	−2	−2	−3	−3	−2	−2	−2	−5	−4	+10	+16
61	6/5	6/5	−½	60	−3	−4	−4	−4	−3	−2	−3	−2	−2	−2	−2	−2	−10	−10	+40	+36
62	6/5	6/5	+½	64	−4	−4	−4	−4	−4	−4	−4	−4	−4	−4	−4	−3	−6	−7	+26	+38
63	6/6	6/5	+2	66	+3	+4	0	+2	+5	+6	+6	+4	0	0	0	0	−10	−8	+100	+70
64	6/6	6/5	0	64	−2	−4	−3	−4	−2	−2	−2	−4	−4	−4	−4	−4	−10	−8	+36	+50
65	6/6	6/6	−½	62	−4	−4	−4	−4	−2	−2	−2	−2	−2	−2	−2	0	−6	−8	+52	+48
66	6/9	6/6	+½	63	−3	−3	−3	−3	−3	−1	−1	−1	−3	−1	−3	−3	−9	−9	+19	+29
67	6/6	6/6	+1	68	−2	−2	−4	−4	0	0	+1	0	−2	−3	−2	−2	−7	−6	+4	+4
68	6/6	6/6	+1½	68	−6	−2	−2	0	+2	+4	0	+3	−4	−4	0	0	−8	−6	+18	+16
69	6/6	6/6	+½	65	−4	−4	−4	−2	−2	−2	+1	+1	−4	−4	−2	−2	−6	−6	+38	+54
70	6/6	6/6	+1	63	0	0	0	−2	−1	−1	−1	0	−1	+1	−2	−4	−9	−9	+60	+60
71	6/5	6/5	+½	62	−4	−5	−4	−4	−4	−4	−4	−4	−4	−4	−4	−4	−7	−6	0	+10
72	6/5	6/5	+1	63	−3	−3	−3	−4	−4	−4	−4	−4	−4	−3	−4	−2	−5	−6	+70	+112
73	6/6	6/6	+2	62	0	0	0	0	0	+2	+4	+1	0	0	0	0	−5	−4	+4	+5
74	6/9	6/9	−3½	67	−10	−12	−10	−10	−6	−4	−2	−6	−8	−10	−8	−10	−12	−12	0	+23
75	6/9	6/5	+3	64	−2	−4	−2	−4	−2	−3	−2	−2	−2	−2	0	−2	−4	−5	+30	+18
76	6/5	6/5	+2½	64	−2	−2	−4	−2	−2	−2	−2	−2	−4	−3	−2	0	−6	−6	+10	+44
77	6/6	6/6	+4	63	−4	−4	−4	−4	−2	0	−2	0	−2	0	0	+2	−8	−5	+50	+44
78	6/5	6/5	+3	61	−4	−4	−4	−4	−2	−2	−2	−2	−2	−2	−2	−2	−8	−8	+50	+48
79	6/9	6/6	0	62	−6	−6	−7	−7	−2	−2	−2	−2	−6	−6	−4	−1	−12	−10	+28	+25
*80	6/9	6/9	+1	60	−2	−4	−4	−4	−2	−2	−2	−1	−4	−2	−2	0	−7	−5	+54	+38
81	6/6	6/6	+1½	63	−4	−4	−4	−4	−3	−4	−4	−4	−4	−4	−2	−3	−7	−7	+54	+54
82	6/6	6/6	+1	64	−4	−4	−4	−4	−4	−4	−4	−4	−6	−4	−5	−6	−8	−6	+20	+38
83	6/5	6/5	+1½	66	−4	−4	−6	−5	−5	−5	−6	−5	−5	0	0	−1	−10	−10	+30	+26
84	6/6	6/6	+2½	66	−2	−2	0	0	+2	0	0	0	0	+1	+2	+2	−6	−4	+28	+36
85	6/5	6/5	+1	61	−2	−2	−2	−1	0	0	0	+1	−5	−5	−4	−4	−8	−6	+20	+26
86	6/9	6/6	+2	62	−6	−6	−5	−5	0	+2	+2	+4	−5	−4	0	0	−2	0	+5	+8
87	6/6	6/6	+1	63	−4	−4	−4	−5	−2	−2	−2	−2	−6	−4	−2	−4	−12	−10	+16	+18
88	6/6	6/5	+2½	59	0	0	0	0	0	0	+2	+2	−4	−2	0	−2	−4	−4	+6	+20
89	6/5	6/5	+½	62	−2	−3	−3	−3	−3	−2	−2	−2	−5	−5	−5	−4	−10	−6	+2	+20
*90	6/6	6/6	0	64	−3	−4	−4	−4	0	−2	+2	+2	−3	−4	+2	+2	−4	−3	+12	+28
91	6/6	6/6	+1½	61	−2	−2	−2	−2	−2	−2	−2	−2	−1	−1	−1	0	−6	−6	+20	+26
92	6/6	6/6	−1½	65	−6	−6	−4	−4	−4	−4	−3	−4	−6	−4	−3	−3	−7	−9	+8	+10
93	6/6	6/6	+½	61	−3	−3	−3	−2	−2	−2	−2	−2	−4	−4	−3	−3	−5	−6	+8	+8
94	6/6	6/6	+1	61	−2	−2	−2	−3	−2	−2	−3	−3	−2	−2	0	0	−8	−8	+17	+54
95	6/5	6/5	0	65	−2	−1	−1	−2	+2	+1	+1	0	−2	−3	+2	+2	−10	−10	+30	+45
96	6/5	6/5	+½	62	−6	−3	−4	−3	0	0	+2	+3	−6	−3	−2	0	−8	−9	+25	+31
97	6/5	6/5	+½	61	−2	−2	−2	−2	0	−1	0	0	−2	−2	0	0	−6	−6	+30	+35
98	6/6	6/5	+1	65	0	+2	−1	−1	−1	−1	−1	−2	−1	0	0	0	−6	−5	+8	+12
99	6/6	6/5	+1½	61	−2	−2	−2	−2	−2	−2	−1	−2	−3	−2	−2	−2	−6	−7	+8	+8
100	6/5	6/5	−1	62	−4	−5	−6	−5	−4	−5	−4	−4	−6	−6	−4	−4	−10	−9	+25	+23

*W.A.A.F. personnel.

Maddox rod and synoptophore readings are in prism dioptres.

AN UNUSUAL CASE OF INTRA-OCULAR HAEMORRHAGE*

BY

D. W. McLEAN

FOLKESTONE

THE condition described here appears to be sufficiently unusual to merit recording. I believe it is by no means unique, but I can find no mention of it in books of reference.

Mrs. E. T., age 43 years, presented herself at an out-patient clinic in September, 1945, complaining that the vision of her left eye had been blurred since the previous evening, and it was found that the pupil was half filled by a bead of bright red blood about three millimetres in diameter. Dilatation revealed the presence of a strand of persistent pupillary membrane, arising from the anterior surface of the iris and stretching roughly horizontally across the pupil. The haemorrhage proceeded from this strand, close to the temporal margin of the pupil and extended forwards in front of the margin. There was no visible haemorrhage elsewhere, and no evidence of a haemorrhagic tendency. No history of trauma could be obtained, but as the patient had been playing with a boisterous child immediately before the onset, the occurrence of a minor injury seemed likely.

Under homatropine the haemorrhage was absorbed completely in seven days leaving the vision unimpaired. The membranous strand remained, and slit-lamp investigation revealed a patent blood vessel in which movement of blood was observed.

ANNOTATIONS

Delay in supply of spectacles

The delay in obtaining spectacles is to-day causing a considerable amount of anxiety to ophthalmologists and a great deal of inconvenience to patients. It seems curious to many that whereas during the war there was a greater lag between the writing of the prescription and the delivery of the spectacles than existed before the war, this interval is now very much longer, extending usually into a considerable number of weeks and sometimes into an appreciable number of months. The Faculty of Ophthalmologists

* Received for publication, January 28, 1946.

has recently taken this matter up and it may be of interest to ophthalmologists to know the causes of the hold-up and the prognosis for the future.

Before the war the production of ophthalmic lenses, both mass-produced lenses and those individually made, was quite sufficient to meet the demands in this country, and, moreover, to supply a very considerable export trade principally to the Dominions. During the war this production was very considerably hampered by the destruction of a large proportion of optical factories, particularly those in the London area, where, unfortunately, many of them were sited. Despite this, however, largely by the enthusiastic work of the employees and partly by drafting into the industry a large number of women emergency war-workers, the supply of lenses during the war was maintained at a level which satisfied the demands of the fighting Services, of our Allies, and also, to a very considerable extent, of the civilian population. It is to be noted in this connection that in order to aid the industry the fighting Services used only flat lenses. To-day the picture has changed considerably, although the demands of the fighting Services are now practically negligible and the quantity of ophthalmic lenses exported has been limited to token consignments to maintain old-established markets. On the one hand a very large proportion of emergency female labour recruited for the war has ceased working. Unfortunately also the individual output per man has fallen very considerably from the high peak attained and maintained by the enthusiasm of the war, a tendency not limited to the optical trade alone. Despite this, however, owing to the whole-hearted effort and internal co-ordination of the industry, the total output of lenses in this country to-day is considerably higher than it was before the war.

On the other hand, the demand for spectacles has increased enormously. It is estimated that there are half a million more people wearing spectacles to-day than there were in 1939. This is perhaps a general growing tendency as the population is gradually becoming educated (rightly or wrongly) in the advantages of spectacles in "saving sight." To a considerable extent it may also be due to the fact that a large number of men who were, for example, labourers or agricultural workers before the war and had never experienced the need of spectacles in their work, but had very considerable refractive errors, were given glasses by the Army in order to increase their visual efficiency; having discovered their advantages these men now retain their glasses. In addition, many of the population were drafted into precision work in factories or to clerical work and for the first time found the necessity for optical aids. Again, the cessation of Woolworth's chain-stores as vendors of spectacles has thrown an increased demand of about a million and a half extra glasses a year on the existing optical trade.

Finally, the acquisition of wealth by the working classes has stimulated many people who before the war were quite happy to wear flat lenses to demand torics, or who used to be content with one pair of spectacles to buy two. It is interesting that while before the war the sale of flats compared to torics was about 50 : 50, the demand is now something like 25 : 75 with the result that the facilities for supplying the former are easy, those for making the latter are strained.

Shortly after the cessation of hostilities there was a sudden and enormous demand all over the country for spectacles, due to the reasons already noted, and also to the fact that a large number of people neglected or did not have the opportunity to renew their spectacles during the six war years. This resulted in the industry being swamped at the end of 1945 and the beginning of 1946 and, since the demand has been fully maintained, it has not yet been able to recover itself. It is estimated however, that as new factories gradually become available and as new employees being trained at present become efficient—a time-consuming process averaging about eighteen months—the position ought to be fairly satisfactory towards the end of 1947. In the interim despite the efforts of the trade and despite the fullest support of the Ministries concerned, a considerable delay is inevitable. To assist in minimising this delay the Faculty of Ophthalmologists has made certain recommendations which will be found below. They are not, of course, compulsorily applicable but there is no doubt that if they are generally applied not only will the individual patients concerned receive their spectacles sooner, in many cases by some weeks, but the problem as a whole will be to some extent relieved. It is hoped that ophthalmologists generally will study them and comply with them in so far as they can until the position becomes satisfactory.

Looking into the future, however, the next problem will be how far the present swollen demand for spectacles will be still increased when they become a "free" issue in a National Health Service. It may well be that increased manufacturing facilities will be able to meet the demand. We can only hope that they will.

The Faculty's recommendations are as follows:—

1. Except in cases of genuine medical urgency, avoid priority orders.

2. Unless there are good reasons for the contrary, avoid prescribing 1/8 powers, and ¼ powers over 4D.

3. In the lower ranges, covering the "stock powers," prescribe *flat* lenses whenever possible, except in the case of bifocals where torics should be ordered.

4. Avoid ordering tinted lenses wherever possible. When ordered, give as alternative tints either Crookes' or Softlite.

5. Where possible, indicate on the prescription that a variation

of ¼D. up or down is permissable. It is suggested that this should be done by the sign " ±0·25 " on the prescription.

6. Avoid prescribing prisms unless really necessary. All prescriptions including a prism, other than those that can be dispensed by decentration, demand special grinding.

On consulting rooms

This room is the centre of the ophthalmologists activities. However far his tentacles may spread by way of nursing homes and country journeys the vast majority of his work is done in his consulting room. For eye work a large room, though not essential, is best. We always liked to have a twenty foot range from the test types to the patient, without any arrangement of mirrors to reflect the image of the letters. Up till now most consulting rooms have been make-shift adaptations of some living room in an oldish house. In the future it is possible that consulting rooms will be built specially for the purpose, but it must be owned that our make-shift methods have worked well. It is a convenience if the consulting room be on the same floor as the waiting room; if a half blind patient has to climb the stairs to the first floor there is always a chance of his stumbling and possibly breaking his neck before the ophthalmologist can record his vision. Furniture may be divided into essentials and luxuries. By the essentials we mean a table and chair for the surgeon, a chair for the patient to sit on while he tells his story and comfortable chairs for any relatives or friends who may come with him. If the floor of the room be of any highly polished material it is unwise to rely on small rugs for the covering. These may easily slip and let the patient down and the ophthalmologist be landed in an action for damages for a broken wrist or other bone. On such floors as teak or parquet a carpet is essential. A wash-hand basin is another indispensable piece of furniture. A book case adds to the look of the room and may be filled with either professional or profane literature. In our case complete sets of the *Transactions* and of this journal should be on the shelves. A few pictures, prints of either Lord Lister or Sir James Paget, or in our own case, Sir William Bowman may be hung on the walls.

Polonius's advice as to raiment is applicable to the furniture; as good as you can afford, " rich but not gaudy." When the writer started in practice he housed for an elder brother a fine copy of a roll-top desk of the Sheraton period, together with an original Bergère chair for his own accommodation. A ribald relative who called on us said that the desk alone was enough to make the fortune of any professional man. It did'nt make our's, for we had to return it to its owner after six months. But the chair adorned our room till a weighty patient, sitting down to write

a cheque, broke through the cane work of the seat, and after its repair we kept it at home for less ponderous visitors.

The impedimenta of our craft are of course essential and are too well known to need enumeration. Among the luxuries one would include a couch or sofa in case any minor surgery has to be performed on a patient in the supine position. Here, we may state our conviction that it is a mistake to turn the consulting room into an operation theatre. Even chalazia are much better attended to in a nursing home and the charge for the temporary use of the theatre for this small operation does not add much to the patient's expenses.

The room should be furnished for what it is:—a professional man's office. If you have any really valuable furniture, or "rare and curious old masters" you can have these in the room if you like, but our own feeling is in favour of keeping these in the more private parts of the house.

Good blinds and curtains are a necessity unless of course a proper dark room has been constructed in a recess.

A man's hobbies are sometimes reflected in his consulting room. A fisherman may have a stuffed carp in a case on the wall, a big-game enthusiast, a tiger skin on the floor. A mountaineer, a picture of the Matterhorn, or a pair of ice axes placed in saltire over the chimney piece. The gentleman who used the writer's consulting room before he came into residence had been a first class pistol shot, and several cases of pistols were there.

ABSTRACTS

MISCELLANEOUS

(1) **Reese, A. B. (New York).—The iridencleisis operation for glaucoma.** *Arch. of Ophthal.*, Vol. XXXIV, No. 5, p. 360, December, 1945.

(1) The technique evolved by **Reese** has as its objects:

1. Cutting of the sphincter muscle.
2. Reaching the periphery of the iris with the radial cut.
3. The absence of the root of the iris over the coloboma.

It is carried out as follows, under retro-ocular novocaine and adrenalin. The point of the keratome engages the conjunctiva 10 mm. from the limbus, and drags it down to within 2 mm. of the limbus, where it incises the sclera, being pushed on until at least ¾ of the blade is within the anterior chamber. The conjunctival opening is enlarged on each side with scissors, the iris grasped at the

pupillary margin with forceps. An assistant holds the iris on the opposite side, and he and the surgeon withdraw it until an iridodialysis is just noted. A radial cut is then made through the iris into the iridodialysis opening, the assistant releases his forceps, and the operator incarcerates each pillar into the corner of the wound with minimum manipulation. The conjunctiva is closed with interrupted silk sutures, and no drops are instilled. It is important for the keratome to enter the anterior chamber in, or close to, the angle, in doing so the point of the keratome may engage the iris, pushing it ahead, and so producing a peripheral dialysis, which is rather an advantage than otherwise. The commonest fault is for the incision to be too far forward. After dividing the iris, the ends should be allowed to curl over, so as to form two tubes lined by pigment, since this is the most advantageous condition for establishing drainage. In order to facilitate this, the lips of the wound may be separated a little, by grasping the conjunctival flap and pulling it forwards at the time of the incarceration. In discussing indications, the author states that iridencleisis is suitable for cases with a peak pressure of 40-50 mm. Hg and a basal pressure of 30 or less. When the pressures are higher, it is advisable to combine it with a sclerectomy. It may be used in secondary glaucoma with inactive iritis, and in buphthalmos. Contra-indictions to the operation are, in the author's opinion, cases in which one or more previous surgical procedures have failed, cases of long standing glaucoma in which the iris has become vascular and atrophic, and in acute glaucoma, for which he has found broad iridectomy to be adequate.

In the discussion following the paper, Minsky gave a useful method of determining the correct site for the scleral incision. The limbus below is focally illuminated, and a brilliant crescent appears above on the sclera at the periphery of the anterior chamber. The point of the keratome should be engaged at the very edge of the crescent, and passed through the sclera in the plane of the iris.

F. A. W-N.

(2) **Fisher, J. A. (Ashbury Park, New Jersey).—Severe laceration of an only eye with recovery of useful sight.** *Amer. Jl. Ophthal.*, Vol. XXIX, p. 1014, 1945.

(2) **Fisher** emphasises the importance of conservative treatment in cases of severely injured eyes. His case illustrates that even an eye with an extensive scleral rupture involving nearly half the globe with iris prolapse, extrusion of the lens and vitreous haemorrhage may recover to 20/50 vision after wound toilet and scleral suturing.

H. B. STALLARD.

(3) **Schultz, A. (M.C.) A.U.S. (Fort Devers, Massachussetts).—Boeck's sarcoid with uveo-parotitis and dacryoadenitis.** *Amer. Jl. Ophthal.*, Vol. XXIX, p. 1010, 1945.

(3) **Schultz** describes a case of Boeck's sarcoidosis with the unusual association of uveoparotitis and dacryoadenitis. He suggests that this corroborates the mounting evidence that uveoparotitis and Mikulicz's syndrome are frequently manifestations of Boeck's sarcoid.

H. B. STALLARD.

BOOK NOTICES

Penicillin—Its Practical Application. General Editor: PROFESSOR SIR ALEXANDER FLEMING, M.B., F.R.C.P., F.R.C.S., F.R.S., Professor of Bacteriology in the University of London, St. Mary's Hospital, London. Pp. 362, 59 illustrations. London: Butterworth and Co. 1946. 30/-.

The purpose of this book is to give an authoritative guide to medical practitioners in the use of penicillin. Under the general editorship of Professor Sir Alexander Fleming a number of authors have contributed chapters concerning the chemistry, pharmacology, pharmacy, and manufacture of penicillin; the bacteriological control of penicillin therapy and its methods of administration. Its clinical use in various specialties is described by experts in these particular branches of medicine and surgery. Professor Sir Alexander Fleming has written an admirable account of the history and development of penicillin and the bacteriological control of penicillin therapy.

Professor Arnold Sorsby has written the chapter on ophthalmology. In this he deals ably with the therapeutic use and dosage of penicillin in certain inflammatory disorders of the eye and its adnexa. Here as elsewhere in this book it is not claimed that the last word has been said on the technique of application and the dosage of penicillin. With the production of purer forms of this valuable agent larger doses may be administered and severe infections such as cavernous sinus thrombosis and endophthalmitis may be controlled and the patient saved from the ravages of such serious disorders. Further laboratory and clinical research is going on and improved methods of using penicillin are becoming evident.

There is much of both scientific and clinical interest in this book which fills a pressing need at the moment. It is certain that it will be widely read. The production and illustrations are good.

Researches on Normal and Defective Colour Vision. By W. D. WRIGHT, A.R.C.S., D.Sc., Reader in Colour Vision, Imperial College of Science and Technology, London. With a Foreword by L. C. MARTIN, D.Sc., Professor of Technical Optics, Imperial College of Science, London. Pp. 383, with 233 illustrations. London: Henry Kimpton. 1946.

The first experiments on colour vision based upon Young's trichromatic theory were made by Helmholtz (1852-3) and Clerk-Maxwell (1855-6). More accurate determinations of the three so-called "sensation curves" were made by König (1883-1901) and by Abney (1886-1913). Even then the experimental conditions were not ideal, and it became evident that new estimates of the trichromatic coefficients and the curves derived from them should be made. The opportunity arose in 1926, when Wright, under the aegis of the Medical Research Council, devised a new trichromatic colorimeter with spectral primaries (1926), subsequently still further refined (1938). His mean results, when correlated with those of previous observers and with the work of Ives (1915, 1923), Guild (1924), Stiles (1939), Walters (1942), Pitt (1944), and others, have provided the most accurate data yet available. In addition they have revealed new features associated with adaptation, discrimination of hue, area of stimulation, retinal location and so on which have raised fascinating new problems when taken into consideration with the physiological researches of Lythgoe, Adrian, Granit, Hartline and others.

Particularly interesting are the measurements on colour adaptation by a binocular matching technique. In this the right eye observed a test stimulus whose appearance was matched against a mixture of three colorimeter stimuli, 0·65μ, 0·53μ, and 0·46μ, as seen in the left eye. The right eye was then adapted to a given stimulus and the effect of adaptation on the appearance of the test stimulus was recorded by matching the test stimulus as seen by the light adapted right eye against the mixed stimuli as seen by the constantly dark-adapted left eye (1934-4).

The apparatus has also been used to measure the characteristics of various forms of defective colour vision by Wright, Pitt, Nelson and others.

The book gives not only a most lucid description of the experimental conditions and results, but also a clear critical analysis of the theoretical principles which underlie the extremely complex phenomena of colour vision. It is sufficient to say that it is a worthy successor to König's Gesammelte Abhandlungen and to Abney's Researches on Colour Vision, and that it is an outstanding landmark in the history of the subject.

CORRESPONDENCE

QUININE AMBLYOPIA

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—In the descriptions given of quinine amblyopia, it is stated that the disc is pale and the vessels greatly constricted. In this connection, an interesting case came under observation in Changi P.O.W. camp. The patient, Sapper W., of the Royal Engineers, was in hospital with jaundice when he developed malaria. He had previously been under observation for his eyes with conjunctivitis and (??) retrobulbar neuritis, due to deficiency disease, but had not been under treatment for it. (He came under my observation first on July 12, 1945).

On July 10, 1945, when his M.T. malaria developed, he was put on quinine, grains 30, daily, for seven days, this being the usual treatment.

On July 11, 1945, he developed very severe headache and also became restless. He was given aspirin, grains 10. During the afternoon he developed tinnitus, and about 5.30 p.m. complete loss of vision with pupils widely dilated.

He was not seen by me until next day at about 11 a.m. When I examined him he had complete amaurosis. His pupils were widely dilated, inactive to light and accommodation. Hippus was present, the media were clear, and the fundi appeared normal. The vessels were of normal calibre. General examination showed no cerebral changes; central nervous system N.A.D.; blood pressure 90/52.

He was given two capsules of amyl nitrite to inhale—acetylcholine was not available in the camp. In the afternoon about 3 o'clock, the patient reported that there was some return of vision, particularly in the right eye. He stated that everything appeared to be blue. Fields were constricted to tube vision.

On July 13, 1945, vision still improving, still blue, and the patient complained of photophobia.

I did not examine him again thoroughly until July 21, 1945. Visual acuity, hand movements only, everything still appeared blue, fields very constricted. He could not distinguish the Ishihara vision charts. Stated that the demonstration chart (red 12 on blue background) appeared as blue figures on a blue circle on light blue background. Examination of fundi showed discs normal, but arteries very constricted and veins reduced in calibre. The arteries left the disc more or less normally, and then tapered away rapidly to mere threads. Otherwise nothing abnormal in the fundi.

On July 24, 1945, pupils were active to light, the blue vision was

still present, and the fundi showed the vessels still very constricted.

On August 19, 1945, his vision had much improved, he could read slowly J.8. His vision was still somewhat blue. The fundi showed the vessels to be much more prominent and the fields fairly full to hand movements.

On September 6, 1945, vision much improved. Blue vision had disappeared, but he noticed what he described as blackness in the later afternoon and at night.

Examination of the eyes showed the discs normal, vessels still much constricted, especially the arteries, and the fields showed peripheral constriction.

He was not seen again after this by me, as we had been relieved by the incoming forces.

I was informed that about 40,000 patients had received treatment for malaria, either primary or relapses, in the camp during our period as P's.O.W. This was the only case of quinine amblyopia which occurred.

I think it is interesting to note that the fundi were normal and vessels normal in calibre 48 hours after the onset of the complete blindness, and only later did the constriction appear, also that the discs appeared normal throughout.

I wish to thank the Director-General of Medical Services, for permission to publish this case.

Yours faithfully,

R. GRAEME ORR.

8, COLLINS STREET,
MELBOURNE.

September 25, 1946.

GRADES OF VISION

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—Having read the article by Mr. Joseph Minton, and noted the suggested grades of vision, I was wondering if the problem of amblyopia in returned P's.O.W. had been considered at all. Observation of, and discussion with, many of them has suggested to me that the disability for certain occupations may be much beyond what is suggested by the ordinary test for visual acuity.

To begin with the standard of 6/6 is for many people, a low one; many can easily attain 6/5 and 6/4. Consequently, those who have been reduced from 6/4 to 6/6 or 6/9 by disease are at a disadvantage.

When the lowered visual acuity is caused not by refractive errors

or interferences in the media, but by areas of scotomata near the fixation point, then the disability may be very much greater. It is not so much a matter of the size of the scotoma, but its position.

If the scotomata lie so that the area affected is to the right of the point looked at (i.e., between the macula and disc) then the patient may have great difficulty in reading fluently, because as his eye travels along the line from left to right, each subsequent word in turn is missing.

If the scotoma is projected so as to lie below the point of gaze, then the patient is inclined to jump down two or more lines when his eyes move to the left to commence a fresh line. Some of these patients had 6/5 and 6/4 vision. Clerical work for these people was difficult, given time they could do it, but not readily.

But when the visual acuity was 6/9 and 6/12 reading became almost impossible. One such patient—an English doctor with 6/12 in each eye—came to the conclusion he would have to give up his occupation as he could not read, except the print were large, and then only slowly.

As regards Naval, Mercantile Marine, and Railway employees, dependent upon seeing lights—white or coloured, at night—even 6/4 vision would be dangerous if the scotomata were present.

In support of this, I give the case of an amateur yachtsman with 6/5 vision in the right and left eye, and an amblyopia due to deficiency disease, who lived in the same quarters with me in Singapore overlooking the Johore Straits, who slept poorly at night and used to spend some of his wakeful hours watching an occulting gas buoy with momentary white flash. He remarked to me that he had been puzzled for some days to be unable to find any fixed period for the light, which was about one mile away.

Actually, the light flashed every three seconds but his scotomata, though small, obscured his vision sufficiently to make him quite unreliable. He was under observation for three years and never was his vision less than 6/5 in each eye. He could read small print comfortably.

Though I think it possible that many P's.O.W. thus affected by scotomata may recover somewhat with time, and in addition, may compensate to some extent for their disability, yet it seems to me that special care may have to be taken, lest in certain occupations they be a menace not only to themselves, but also to others.

Yours faithfully,

R. GRAEME ORR.

8, COLLINS STREET,
MELBOURNE.

September 23, 1946.

OCULAR LESIONS IN INTERNEES AT A CIVILIAN INTERNMENT CAMP AT HONG KONG

To the Editors of THE BRITISH JOURNAL OF OPHTHALMOLOGY.

DEAR SIRS,—Dr. Talbot's observations upon ocular manifestations among civilian internees in Hong Kong differ materially from my own and those of my ophthalmic colleague in military P.O.W. camps in the same area, and also from those of other British and Australian specialists who have studied them elsewhere in Japanese occupied territory in the Far East.

That the central scotoma which was so characteristic elsewhere should have been "surprisingly exceptional" is indeed surprising and requires explanation.

I would suggest that this may be found in the methods used in the investigation of the fields. Dr. Talbot gives us no indication of these beyond the statement that he is indebted to a Hong Kong Government official who "carried out practically all the examinations of the fields." We are not told what qualifications he possessed for this task beyond "immense patience."

Scotometry is an art which is often somewhat indifferently practised by ophthalmic specialists themselves.

More convincing evidence of the accuracy of the field examinations is necessary before admitting the validity of any deductions drawn from them, particularly when they conflict with those of experienced observers in similar situations.

Yours faithfully,

JOHN DURRAN, F.R.C.S.E., D.O. OXON.
Chief Assistant, Ophthalmic Dept.,
Leicester Royal Infirmary.

LEICESTER,
November 7, 1946

OBITUARY

HENRY HANNA

IT is with deep regret that we record the sudden death of Mr. Henry Hanna on September 28, 1946, at his residence 30, University Square, Belfast.

Born in 1874 he received his early education at Belfast Royal Academy while his university career was spent between Queen's College, Belfast, and St. John's College, Cambridge. The old Royal University of Ireland conferred upon him a Bachelor of

Arts degree in 1894 and a Master of Arts, Bachelor of Science degree in 1896. In the following years he worked at Cambridge University but in due course returned to Ireland to commence a medical career. His undergraduate studies finished when he was awarded his Bachelor of Medicine degree in 1903.

Having completed a resident medical officer appointment in the newly built Royal Victoria Hospital, Belfast, and a period of demonstrating in the department of anatomy, he proceeded to Vienna to study at their eye, ear and throat clinics. Returning to Belfast he commenced to practise in his chosen speciality and in due course became head of the department in the Royal Victoria Hospital and also the Belfast City Hospital.

His colleagues honoured him with many of their highest offices as he was President of the Irish Ophthalmological Society, President of the Ulster Medical Society, and also presided over his special section at the British Medical Association meeting in Belfast in 1937.

His interests outside medicine were varied. He was often to be seen on the golf links, but perhaps his greatest joy was fishing in a quiet stream. He had a very genuine appreciation of art and for many years was a most enthusiastic collector. His collection includes many valuable pictures and porcelain as well as a beautiful selection of old Irish glass.

Henry Hanna retired from the active staff of the Royal Victoria Hospital just before the outbreak of war in 1939. His period of retirement was, however, short-lived for as the younger men proceeded to join the fighting forces he was asked to return to his old hospital and carry on. He and his contemporaries did a noble job, for without their help our hospitals could not have carried out their tasks. We of a younger generation are very grateful to them for their unselfish service in our hour of need.

To his wife and two daughters we extend our sincere sympathy.

NOTES

Honour MR. R. R. JAMES has been elected an honorary member of the Oxford Ophthalmological Congress.

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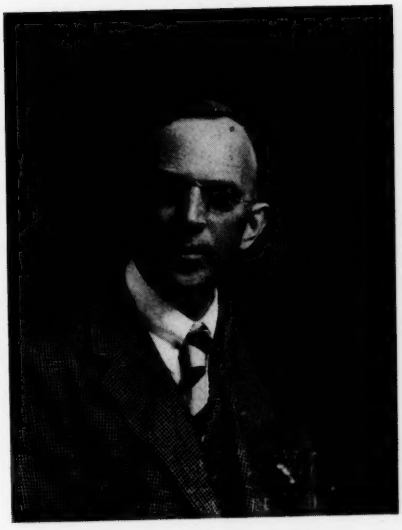
I.A.M.C. Journal A copy of the Indian Army Medical Corps Journal (Vol. II. No. 2, June, 1945), has been sent to us. The Journal is published twice yearly and is edited by Col. D. R. Thapar, C.I.E., O.B.E., M.D. The contents are of general interest, and there is little bearing on ophthalmology in this



HENRY HANNA, 1874-1946.

issue. Papers on blood groups; psychiatric casualties amongst I.O.R.'s; treatment of cholera and acute gastro-enteritis; medical stores; repatriated allied prisoners of war and internees, together with an editorial, administrative notes, "Who's Where," with Honours and awards and some entertaining reminiscences of an earlier day complete the number, which is introduced by a stirring call to officers and men of the corps by the Director of Medical Services in India, Lieut.-General Trefiry O. Thompson, C.B., C.B.E., K.H.P., who, until recently, was Medical Adviser to the Supreme Allied Commander, South East Asia.

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III Pan-American Congress of Ophthalmology

THE Third Pan American Congress of Ophthalmology will take place at Havana, Cuba, from Monday 23rd to Saturday 28th February, 1948. The programme includes the following sections: (*a*) Official subjects; (*b*) Sessions of Research and Social Works; (*c*) Scientific Expositions; (*d*) Instruction Courses; (*e*) Cinematograph Exposition.

Papers should be in the hands of the Local Committee by February 23, 1947, for the Official subjects. No changes can be made in the programme after August 23, 1947. Papers should be accompanied by an official summary of 300 to 500 words in the Official subjects and from 200 to 300 words in the free communications. The Local Committee will send out informative bulletins every three months. Those who wish to attend the congress should send their names to Dr. Tomas R. Yanes, P.O. Box 970, Havana, Cuba, from whom information as to subscriptions, etc., can be obtained.

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Sociedad Cubana de Oftalmologia. Officers, 1946-48

President, Dr. Lorenzo Comas; *Vice-Presidents*, Dr. Miguel Méry and Dr. Rodolfo Hernández; *Secretary*, Dr. Oscar Horstmann; *Vice-Secretary*, Dr. Tomás R. Yanes; *Treasurer*, Dr. Cándido Durán; *Vice-Treasurer*, Dr. José Ruíz Velazco.

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The Middle East Optical Practitioners' Group. Spring Session, 1946

VOL. II contains papers read by eye surgeons and opticians to the above society at the Spring session, 1946. The subjects of the papers include glaucoma, cataract, diseases of the optic nerve, psychology, protective lenses, the innervation and action of the ocular muscles and the nature of light.

This session closed the meetings and Transactions of the Middle East Optical Practitioners' Group which it is evident has been a happy enterprise.

A Services Club

AN Inaugural Dinner of surgeons who served with the Army in the Central Mediterranean Forces was recently held at the Royal College of Surgeons of England under the Chairmanship of Sir Alfred Webb Johnson. It was suggested by Mr. Harold Edwards that a Surgical Club should be formed to perpetuate the happy associations which had been formed in the Army.

All specialist, graded and trainee ophthalmologists who served with the Central Mediterranean Forces, are invited to become members of this Club, and to do so they should send their names and present addresses to Mr. Harold Rogers at St. Bartholomew's Hospital, who is acting as Hon. Secretary. In due course each ophthalmologist will receive an individual communication.

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Corrigendum

THE photograph in Fig. 1 (certain operations on the superior oblique, p. 678), was stated by the letterpress to describe the patient looking to the right. The wrong photograph was sent in error, and the correct one is given below. In an attempt to correct this (after the proofs had gone in), the letterpress was mistakenly altered on p. 677 to conform with the incorrect picture. The vertical diplopia in this case was in fact accentuated on looking to the *right*, p. 677, line 9, and the deficiency of movement was downward and to the *right* not to the left, p. 677, line 24—in other words, that of a characteristic left superior oblique paralysis.

FIG. 1.